



# A SYNOPSIS OF ANÆSTHESIA

BY

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TO MY WILL



Eternal vigilance is the price of safety

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Relief from pain is purchased always at a price  
The price in both morbidity and mortality does  
not greatly differ whatever the agent or agents  
used —R M WATERS

---

The duty of the anæsthetist towards his patient  
is to take care

---

While the anæsthetist's chief function is to  
prevent and alleviate pain his primary responsibility  
is to maintain respiration

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*Primum non nocere*— First of all do no harm

## PREFACE TO THE FOURTH EDITION

IN the wide field of Anæsthesia it is astonishing what changes in theory and practice can take place in the brief space of five years. Such changes have called for a major revision of this small book in order to bring it reasonably up to date.

This edition differs from the previous one in having many new figures and my thanks are due to the various manufacturers for lending blocks.

A very complete revision has been carried out and the alterations and additions are so extensive involving almost every page that it is not possible to give them in detail here. Two new chapters have also been added—one on the phenothiazine derivatives and the other on induced hypothermia. There is a new section dealing with halothane (fluothane).

The author has consulted many papers in the anæsthetic and general surgical and medical literature and references are given for most of the new work mentioned. He would also like to express his debt to the authors of the many text books to which he has referred.

As the academic demands on the young anæsthetist become greater his need for consulting original texts and articles increases. It is hoped that the new edition of this small book will serve as a stimulant to wider reading and will help to recapitulate knowledge gained in a broader field.

This short introduction suffices I hope to explain the nature of the book to which it stands preface. There remains only the pleasant duty of thanking the publishers for their constant help, consideration and co-operation.

*January, 1959*

J ALFRED LEE

## FROM THE PREFACE TO THE FIRST EDITION

THIS book is not designed to take the place of the larger textbooks of anæsthesia and analgesia. It is a summary of current teaching and practice and it is hoped that it will serve the student, the resident anæsthetist, the practitioner and the candidate studying for the Diploma in Anæsthetics as a ready source of reference and a quick means of revision.

*January 1947*

J ALFRED LEE



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# A SYNOPSIS OF ANÆSTHESIA

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## CHAPTER I

### NOTES ON THE HISTORY OF ANÆSTHESIA

**Joseph Black** (1733-1790) — The discoverer of carbon dioxide or fixed air. Born at Bordeaux France of Irish Scottish parentage and educated at Belfast and at the Universities of Edinburgh and Glasgow. Became Professor of Anatomy and Lecturer in Chemistry in the University of Glasgow and later Lecturer in Chemistry in Edinburgh. Was all this time a practising physician. In 1754 described fixed air as he called carbon dioxide and described its method of identification by lime water. He proved that the gas produced in respiration during the fermentation of wine (this had been described by von Helmont) during combustion of charcoal in air and that liberated from chalk by heat and acids was one and the same. He showed it to be toxic to animals and that it can be absorbed by alkalis. facts made use of by anesthetists to-day.

**Joseph Priestley** (1733-1804) — The discoverer of oxygen in 1771 and of nitrous oxide in 1772. He was also the first to describe fluorine sulphur dioxide and methane.

Born in Yorkshire and became a Unitarian minister. After an interregnum as secretary to Lord Shelburn returned to take charge of a church in Birmingham. Here he became intimate with Erasmus Darwin James Watt and Wm Murdock the inventor of gas lighting. Because of his advanced political views and his sympathies with the French Revolution his house was beaten up by the mob and he was forced to flee the country. In 1794 he emigrated to Pennsylvania where in addition to carrying on with his scientific studies he was a farmer. He died at the age of 70.

**Humphrey Davy** (1778-1829) — Born in Cornwall the son of a wood carver. Became apprenticed to J B Borlase surgeon of Penzance. At the age of 17 he experimented with nitrous oxide and the effects of its inhalation. In 1798 Davy became superintendent of Thomas Beddoes's Pneumatic Institute in Clifton Bristol and in the following year published his book *Researches Chemical and Philosophical Chiefly Concerning Nitrous Oxide*. In this Davy suggested that nitrous oxide inhalations might be used to relieve the pain of surgical operations. A nitrous oxide container was made by James Watt in 1799 to assist this research. In later life Davy became famous. He invented the miner's safety lamp was created a baronet and was elected President of the Royal Society in 1820.

**Michael Faraday (1791-1867)** — Said to be the first man to note the narcotic effects of ether vapour but this is doubtful. Born at Newington Butts of poor parents he became a paper boy and later graduated to book binding during which occupation he made his first contact with chemical literature. Deciding to become a chemist he obtained the post of laboratory assistant to Humphrey Davy at the Royal Institution in 1813 and a little later accompanied him on an extensive tour in Europe. Became Director of Laboratory of Royal Institution in 1825. His great ability soon threatened to rival that of his master who became jealous. Later Faraday too achieved world wide fame. Fulleren Professor of Chemistry 1833. Discovered benzene. His observations on ether were published in 1818 in *The Quarterly Journal of Science and Arts*.

**Henry Hill Hickman (1800-1830)** — Medical education received in Edinburgh. He settled in practice in Ludlow. While doing a locum at Shifnal in Shropshire his interest in gas therapy was aroused as the village was the birthplace of Thomas Beddoes. Familiarizing himself with the pioneer work of Davy, Priestley and Faraday Hickman returned to Ludlow and commenced experiments on animals. He was able to perform surgical operations painlessly on them by causing them to inhale carbon dioxide. This was the first work on surgical anaesthesia induced by inhaling a gas. His results were published in a paper *A Letter on Suspended Animation* in 1814 but attracted no attention from scientific men in England. Even Sir Humphrey Davy who was approached by Hickman friend T. A. Knight F.R.S. showed no interest. Charles N. of France was appealed to in 1818 and the French Academy of Medicine agreed to investigate Hickman's results but nothing came of the matter. Baron Larrey one of Napoleon's surgeons however gave Hickman some encouragement. Hickman died prematurely aged 29 and was buried in Bromfield churchyard.

**Horace Wells (1815-1848)** — In 1844 Gardner Q. Colton a travelling lecturer in chemistry gave a demonstration of the effects of inhaling nitrous oxide at Hartford Connecticut. Horace Wells a local dentist was present and noticed that a young shop assistant while under the influence of the gas banged his shin and made it bleed but stated that he experienced no pain. Wells persuaded Colton to try the gas during a dental extraction and on the following day Dec 11 1844 the experiment was carried out with Colton as anaesthetist, Wells as dentist and Wells as patient. It was a big success. A new era in tooth pulling according to Wells. Wells learnt from Colton the method of manufacture of nitrous oxide and used it in his dental practice. Later in the year he went to Boston to interest a larger audience in his discovery. He demonstrated the method to the students of Harvard Medical School but the patient complained of pain. The affair was a fiasco and Wells was hushed out of the room as a fraud. He returned to Hartford and continued to use the gas but the introduction

of ether gradually ousted the use of nitrous oxide. Wells gave up dentistry, travelled round the country with a troop of performing canines, and was incarcerated in jail after bespattering a New York prostitute with sulphuric acid. He committed suicide.

Colt reintroduced the use of nitrous oxide in dentistry in 1863 at New Haven.

**William Thomas Green Morton (1819-1868)** — Morton deserves the chief credit for the introduction of ether as an anæsthetic agent, although W. J. Clark of Rochester, New York gave ether for a dental extraction in 1841, and Crawford Williamson Long (1815-1875) removed a tumour from the neck of J. M. Venable quite painlessly in Jefferson County, Georgia a few months after Clark's experiment. By the time (1846) that Long reported his work Morton's fame was well established.

Morton, born at Charlton, Worcester County, Massachusetts, was a dentist who became a student and later a partner of Wells at Hartford. He separated from Wells and becoming a medical student in Boston was present when Wells failed to satisfy the audience as to the efficiency of nitrous oxide. Charles A. Jackson, one of Morton's lecturers at Harvard, suggested that ether could be used as a surface analgesic in dentistry. Morton, however, went further: he experimented on dogs to find out the effect of giving ether vapour by inhalation. Impressed with the results, he gave the vapour to Eben Frost for the removal of a tooth on Sept. 30, 1846. The operation was painless. After gaining further experience and while still a medical student, Morton gave a demonstration at the Massachusetts General Hospital on Oct. 16, 1846, when Dr. J. C. Warren removed a tumour from the jaw of his patient, Gilbert Abbott, without producing any pain. This success gained him the support of Warren and also of Jacob Bigelow, Professor of Materia Medica. Much wrangling occurred between Morton and Jackson as to who should be given credit for the discovery. Morton three times petitioned the U.S. Congress and even obtained an interview with the President, but he was never in his lifetime officially recognized as the pioneer of ether anaesthesia. Time later vindicated his claim. He spent his later years farming and died of cerebral hæmorrhage, a disappointed man. His agent, which he tried to patent under the name *Lætheon*, became widely used. It was given in London and Paris in 1846. Robert Liston was the first surgeon to operate under ether in England: this was at University College Hospital on Dec. 21, 1846, using Squire's inhaler.

The name *anæsthesia* was suggested by Oliver Wendell Holmes, but had been used by Plato in 400 B.C. to denote absence of feelings in a philosophical sense and also by Dioscorides in the first century A.D. to denote absence of physical sensation (Armstrong Davison).

Ether became known in England through a letter written by Henry Bigelow, Jacob's son, to his friend Dr. Boott, who gave the first ether anæsthetic dental case two days before Liston's first use of it. Malgaigne was first man to use ether in France on Jan. 12, 1847.



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- 1818 Faraday is said to have discovered narcotic action of ether vapour ✓
- 1822 Magenlie proved that while anterior spinal roots are motor posterior roots are sensory
- 1824 Hickman carried out operations on animals under carbon dioxide with freedom from pain ✓
- 1825 Cla. Waterton (1752-1845) published his *Habits & Drings in South America* which contained an account of the actions of curare
- ✓ 1831 Chloroform discovered independently by von Liebig, Guthrie and Soubeiran  
Atropine prepared from *Atropa belladonna* by Mein and by Geiger and Hesse
- 1832 Thomas Aitchison Latta used intravenous saline in the treatment of circulatory collapse in cholera (not in surgical shock)
- 1834 Alex. Dumas (1800-1854) described chemical composition of and gave name to chloroform
- ✓ 1842 Ether given by W. I. Clark and by Crawford W. Long in the U.S.  
Flourens first isolated respiratory centre in medulla
- 1844 Horace Wells introduced nitrous oxide inhalation to produce anaesthesia during dental extraction ✓
- ✓ 1846 Francis Rynd of Dublin invented hyperdermic trocar  
Wm. T. G. Morton successfully demonstrated the anæsthetic properties of ether Oct. 16. The word anaesthesia was suggested by Oliver Wendell Holmes for Morton's etherisation  
First surgical operation performed in England under ether anaesthesia by Robert Liston Dec. 21
- 1847 Flourens described anæsthetic properties of chloroform and ethyl chloride vapour in animals
- ✓ James Y. Simpson introduced chloroform into clinical work to ease pains of labour  
John Snow published his book *On the Inhalation of Ether*  
Deaths from ether reported from Grantham and Colchester
- ✓ 1848 Hannah Greener aged 15 died from chloroform administered by Dr. Meggison Jan. 28—the first recorded case—at Winlayton Co. Durham  
Heyfelder first used ethyl chloride in humans
- 1851 Pravaz of Lyons invented hypodermic syringe
- 1853 John Snow gave chloroform analgesia to Queen Victoria at birth of Prince Leopold hence chloroform *à la reine*  
Invention of hypodermic syringe and needle by Alexander Wood of Edinburgh to enable morphine to be deposited at the actual seat of pain or near the nerves supplying the painful area
- 1854 Wm. Gairdner of Glasgow differentiated between post-operative pneumonia and pulmonary collapse the latter due to bronchial obstruction
- 1855 Gaedicke of Germany isolated cocaine from coca plant
- ✓ 1858 Publication of John Snow's book *On Chloroform and Other Anæsthetics*
- 1860 Nieman purified the alkaloid which Gaedicke had isolated from coca leaves. He named it cocaine

**Sir Frederick Hewitt (1857-1916)** — Educated at Merchant Taylors School Christ's College Cambridge and St George's Hospital London where he was a distinguished student. Became an anæsthetist as defective eyesight prevented his becoming a consulting physician and was appointed to Charing Cross Hospital in this capacity in 1884 the National Dental Hospital in 1885 and the London Hospital in 1886. In 1902 became physician anæsthetist to his old teaching hospital St George's. He emphasized that nitrous oxide anæsthesia is possible without asphyxia and that chloroform is specially dangerous during induction. Hewitt modified Junker's chloroform bottle and redesigned Clover's inhaler enlarging the bore of the central tube (as suggested by Wilson Smith in 1901) and arranging for its rotation within the ether reservoir. He devised a dental prop and also an airway (1908) and wrote a popular text book (1893) on anæsthesia (*Anæsthetics and their Administration*) the fifth edition of which appeared in 1922. He strongly advocated better teaching of anæsthetics to medical students. Hewitt invented the first practical machine for giving nitrous oxide and oxygen in fixed proportions in 1887 and the years following. In 1911 he was knighted. Administered an anæsthetic to Edward VII for his appendix operation in 1902.

### IMPORTANT DATES IN THE HISTORY OF ANÆSTHESIA

- ✓ 1516 Curare South American arrow poison described by Peter Martyr Angherius
- ✓ 1540 Valerius Cordus (1515-1544) synthesized sweet vitriol (ether) possibly aided by Paracelsus (1493-1541)
- 1628 Wm Harvey (1578-1657) described the circulation of the blood
- ✓ 1656 First intravenous injection of a drug (tincture of opium) into an animal (a dog) by Sir Christopher Wren and Robert Boyle
- 1660 Boyle enunciated his law of the relationship of the volume and pressure of a gas
- 1665 Richard Lower (1631-1691) transfused blood from one animal to another
- 1754 Carbon dioxide discovered by Von Helmont and isolated by Black
- ✓ 1771 Discovery of oxygen by Priestley and Scheele independently
- 1772 Priestley discovered nitrous oxide
- 1787 Charles's law showing relationship of volume to temperature of a gas
- ✓ 1788 Chas.kite of Gravesend used first endotracheal tube
- 1792 Frobenius a German named sweet vitriol ether
- 1800 Discovery of analgesic properties of nitrous oxide by Davy
- 1806 Isolation of morphine from opium by Serturner (1743-1841)
- 1807 Baron Larrey performed painless amputations using ice on the battlefield
- 1816 René Laennec (1781-1826) invented stethoscope

- 1932 Langton Hewer's *Recent Advances in Anaesthesia* appeared  
 Celfan and Bell of the University of Alberta first used diethyl  
 ether in anaesthesia Celfan acting as the patient
- 1933 Minnitt of Liverpool designed his machine for the self  
 administration of  $N_2O$  and air in labour
- 1934 Guedel described his airway
- 1934 Waters and associates reported on the clinical use of cyclopropane
- ✓ 1934 Allen introduced thiopentone
- 1935 First examination for D.A. held
- 1937 Guedel's *Inhalation Anaesthesia* published
- 1939 Icthadine synthesized by Schumann and Isleb
- ✓ 1940 *Anaesthesiol* 31 first published
- 1941 Trichlorethylene advocated by Langton Hewer and Hadfield
- 1942 Allen reported his work on refrigeration analgesia
- ✓ Griffith and Johnson of Montreal used curare in anaesthesia
- Hingson and Edwards advocated their technique of continuous  
 caudal analgesia
- ✓ 1943 Macintosh described his curved laryngoscope
- 1946 The journal *Anaesthesia* appeared
- 1948 First use of hypotensive anaesthesia by Griffiths and Gillies
- 1949 Fenta and hexamethonium described by Paton and Zaimis
- Short acting muscle relaxants described by Bovet and used  
 clinically two years later in Italy and Sweden
- ✓ 1950 Induced hypothermia in cardiac surgery described by W. G.  
 Bigelow and his colleagues from Toronto

(See scholarly articles by M. H. Armstrong Davison *Brit J Anaes*  
 1957 29 291 and 575 1958 30 142)

## CHAPTER II

### SOME ANATOMICAL AND PHYSIOLOGICAL NOTES

**Respiration** — Defined as the gaseous interchange between an organism and its environment. Oxygen is absorbed and carbon dioxide excreted. External respiration takes place between the alveoli and the capillaries. Internal respiration occurs in the tissue cells.

Pulmonary respiration has two phases. (1) A ventilatory phase or alternating movement of air between the atmosphere and the lung alveoli. (2) An exchange phase an interchange of oxygen and carbon dioxide between the alveolar air and the lung capillaries.

**Nasal Cavities** — Immediately inside the nostril is the nasal vestibule lined by skin giving rise to hairs for coarse filtration and sebaceous glands. The *limen nasi*, a ridge separates the vestibule from the nasal cavity proper which is lined by mucous membrane and extends from before backwards. Thus anteroposterior direction

Important Dates *continued*

- 1911 Goodman Levy proved that chloroform can cause death (from ventricular fibrillation) in light anæsthesia  
Phenobarbitone (luminol) synthesized
- 1912 Boothby and Cotton introduced a sight feed gas and oxygen flow meter  
Kelly was first to use insufflation intratracheal anæsthesia in England  
A. Læwen used curare to produce relaxation
- 1913 Denis was first to describe transsacral analgesia  
✓ Gwathmey introduced rectal oil ether
- 1914 Hustin of Belgium was first to use citrate in blood transfusion  
Gwathmey's *Anæsthesia* published
- 1915 Use of carbon dioxide absorption in animals by Dennis Jackson of Cincinnati
- 1916 Shipway introduced his warm ether apparatus
- 1917 Edmund Boyle described his portable  $N_2O$  and  $O_2$  apparatus  
the chloroform bottle was added in 1920  
Avertin described by Eicholtz and used clinically by Butzengeiger in 1926
- 1920 Guedel's first paper on signs of anæsthesia  
Magill and Rowbotham developed endotracheal anæsthesia
- 1921 Extradural analgesia described by Pagés of Spain  
Carbon dioxide became common in anæsthetic practice following the work of Henderson and Haldane
- 1922 *Current Researches in Anæsthesia and Analgesia* appeared of August  
Goodman Levy's *Chloroform Anæsthesia* published  
First Meeting of Section of Anæsthesia at Annual Meeting in B.M.A.
- 1923 Ethylene introduced by Luckhardt  
Carbon dioxide absorption used in man by Waters  
*British Journal of Anæsthesia* appeared
- 1927 Pitkin introduced spinocain and popularized spinal analgesia  
Ocherblad and Dillon used ephedrine in spinal analgesia  
Pernocton used in Germany by F. Bumm
- 1928 Introduction of circle method of carbon dioxide absorption by Brian Sword  
Lucas and Henderson proved that cyclopropane had anæsthetic properties  
I. A. Magill introduced blind nasal intubation
- 1929 Sodium amytal used by Zervas the first use of rapidly acting barbiturates in anæsthesia given into a vein
- 1930 Waters introduced cyclopropane into clinical practice  
Nembutal and percamne described  
Leake and Chen discovered anæsthetic properties of divinyl ether
- 1931 Dogliotti re introduced extradural analgesia in Italy
- 1932 Weese Scharpf and Rheinoff were the first to use hexo barbitone (evipan)

- 3 The long and short sphenopalatine nerves (from sphenopalatine ganglion)
- 4 The nasal branches of the greater palatine nerve (from the sphenopalatine ganglion)
- 5 The olfactory nerve

**Larynx.**—The organ of voice connecting the pharynx with the trachea. It extends from the root of the tongue to the trachea (Fig 1)

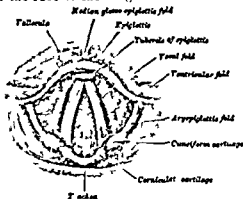


Fig 1.—A laryngoscopic view of the interior of the larynx.  
(From Gray's Anatomy by kind permission of Professor T B Johnston)

It is opposite the third fourth fifth and sixth cervical vertebræ higher in children and in females. The average length is 44 mm in males 36 mm in females its transverse diameter 43 mm in males 41 mm in females its anterior posterior diameter averages 36 mm in males and 26 mm in females. It is covered by the depressor muscles of the hyoid bone by the thyroid gland and by the cricothyroid muscles. Composed of the following cartilages joined together by ligaments thyroid cricoid two arytenoid two corniculate (Santorini) two cuneiform (Wrisberg) and the epiglottis.

The cavity of the larynx extends from the superior laryngeal aperture to the lower border of the cricoid cartilage when it is continuous with the trachea. The piriform fossa is a recess on each side bounded by the aryepiglottic fold internally and the thyroid cartilage and thyrohyoid membrane externally beneath its mucosa lie twigs of the internal laryngeal nerve which are blocked when local analgesic solutions are applied to this area. The depression between the dorsum of the tongue and the epiglottis is divided into two valleculæ by the glosso epiglottic fold. The epiglottis is not essential for swallowing breathing or phonation.

The entrance to the larynx or superior laryngeal aperture is wider in front than behind and slopes downwards and backwards. Bounded anteriorly by the epiglottis laterally by the aryepiglottic folds containing the two small nodules on each side the cartilages of Wrisberg (cuneiform—anterior) and Santorini

*Nasal Cavities continued*

must be remembered when passing nasopharyngeal and nasotracheal tubes. The upper part of the nasal cavity is olfactory the lower part respiratory in function. On the lateral walls are the superior middle and inferior conchæ or turbinate bones with a meatus beneath each.

**OPENINGS INTO THE NOSE —**

- a* The sphenoidal sinus opens into the spheno-ethmoidal recess above the superior turbinate
- b* The posterior ethmoidal cells open into the anterior part of the superior meatus
- c* The anterior and middle ethmoidal cells open into the middle meatus
- d* The frontal sinus opens into the middle meatus
- e* The antrum of Highmore (maxillary sinus) opens into the middle meatus
- f* The nasolacrimal duct opens into the inferior meatus
- g* The Eustachian tube opens from the nasopharynx just behind the inferior turbinate. Nasal tubes have been blamed for originating infection in the accessory nasal sinuses and the middle ear. If such infections are established nasal intubation may be contra indicated.

Mucosa covering the turbinates is capable of great vascular engorgement during infection and allergic reactions and following paralysis of the sympathetic chain in the neck (Guttman's sign) with production of narrowing of the nasal cavities. Vascularity is decreased by the application of adrenaline nor adrenaline cocaine ephedrine and benzedrine also by cold air.

The nasal septum is formed by the perpendicular plate of the ethmoid by the vomer and by the septal cartilage.

Narrowing of the nasal cavities may be (a) Congenital (b) Due to bony deformities e.g. nasal spurs deviated septum (c) Due to engorgement of the mucosa. Obstruction may be caused by foreign bodies blood clot mucus. The degree of patency can be assessed by (a) Inspection with a headlamp and speculum (b) Getting the patient to breathe deeply through the nose while occluding one nostril at a time.

The nasal cavities open into the nasopharynx which is continuous with the oropharynx. Food and air pass through the oropharynx. When food passes the superior laryngeal aperture the latter is reflexly closed while inspiration is inhibited.

Squamous epithelium lines the oropharynx ciliated epithelium lines the respiratory part of the nasal cavities and the nasopharynx. The nerve supply is from the first and second divisions of the fifth cranial nerve via the sphenopalatine ganglion (Meckel) and the nasal branch of the ophthalmic division. The olfactory nerves supply the upper part of the nasal cavity.

The nerve supply of the nasal cavity —

- 1 The anterior ethmoidal (from nasociliary nerve)
- 2 The anterior superior dental nerve (from the maxillary nerve)

the superior thyroid vessels during thyroidectomy such injury causing temporary huskiness of voice. Injury to the recurrent laryngeal nerve on one side causes permanent huskiness with fixation of the cord midway between abduction and adduction—the cadaveric position. With bilateral recurrent laryngeal nerve paralysis speech is impossible and both cords remain motionless. The recurrent laryngeal branch supplies the remaining intrinsic muscles and the mucosa below the cords. The superior aspect of the epiglottis is supplied by the glossopharyngeal nerves. Its stimulation as by Macintosh's laryngoscope does not therefore produce laryngospasm. The inferior surface of the epiglottis gets its nerve supply from the internal laryngeal.

1. Incomplete paralysis of a recurrent laryngeal nerve causes paralysis of abductor before that of adductor muscles and results in respiratory distress from adduction of cords when bilateral.
2. Complete paralysis of recurrent nerve inactivates both abductor and adductor muscles. The tensing action of the cricothyroid muscles maintains cords in adduction.
3. Paralysis of both recurrent and superior laryngeal nerves together produces the cadaveric position.

Arteries are the laryngeal branches of the superior and inferior thyroid arteries.

**Trachea.**—Length about 10–11 cm. It commences at the level of the sixth cervical vertebra and ends by dividing into the two bronchi at the carina level of the fifth dorsal vertebra. Anteriorly this corresponds with the junction of the body and manubrium sterni—the angle of Louis. In children carina is on a level with third costal cartilage. The diameter of the trachea is about  $\frac{1}{2}$ –1 in. (1.5–2 cm.) much smaller in the child e.g. 3 mm during first year of life and thereafter the diameter in mm. corresponds to the age in years.

Abnormal narrowing of the trachea in its middle third making it difficult to introduce a tube of adequate size has been reported.\*

**Right Bronchus**—Shorter (1 in.—2.5 cm.) and more in line with the trachea than the left bronchus. enters the right lung opposite the fifth thoracic vertebra. greater in diameter than left bronchus—hence a long tube or a foreign body passes more easily into it than into the left bronchus. The main upper lobe bronchus given off within  $\frac{1}{2}$  in. of the commencement arises above the right pulmonary artery and was accordingly called the eparterial bronchus. The opening is on a level with the carina. The right bronchus leaves the trachea at an angle of 25° from the vertical.

**Left Bronchus**—Narrower but longer than the above. Length before dividing into upper and lower lobe bronchi 2 in. (5 cm.). The aorta arches over it and it enters the left lung opposite the sixth thoracic vertebra. It leaves the trachea at an angle of about 45° from the vertical. In children under 3 years the right and left main bronchi branch from the trachea at equal angles †

Stewart S. and Finkerton H. H. *Br J Anaesth* 1955 27 492

† Adrian J. and Groggs I. *Anesthesiology* 1954 15 466



## Larynx continued

(corniculate—posterior) posteriorly, by the arytenoids. This view is seen by laryngoscope. The vestibule of the larynx is the superior part of the cavity of the larynx and extends from the aryepiglottic folds to the vestibular folds. Each of the latter is a ridge formed by the vestibular ligament and extends from the angle of the thyroid cartilage anteriorly backwards along the side cavity of the larynx to the cuneiform cartilage. The vestibular folds are the false cords; the space between them is the rima vestibuli while a depression on the side wall of the larynx between the vestibular fold and the vocal fold (false and true cords) is the saccule of the larynx.

The vocal cords (folds) stretch from the thyroid cartilage anteriorly to the arytenoid cartilage of the corresponding side posteriorly. The space between the cords is the glottis. It is bounded in front by the intermembranous part of the cords—the vocal folds behind by the intercartilaginous part. The glottis is the narrowest part of the larynx and measures about one inch from front to back less in females. Its shape and width vary with phonation and respiration and the tone of the muscles controlling it. When these are spastic the glottis is obliterated. The intrinsic muscles are arranged in two functional groups one controlling size the other controlling tension of the cords. The lowest part of the larynx extends from the vocal folds or cords to the cricoid cartilage. The mucosa of the upper part of the larynx is lined by squamous cells like the oropharynx the part above the cords by ciliated epithelium the cords are covered by a thin layer of mucosa closely adherent to them white in colour. The lower larynx is lined by ciliated epithelium with mucous glands and goblet cells.

Intubation has been held responsible for the causation of small innocent tumours of the cords\*. Traumatism in this area can result in dysphagia dysphonia and a husky voice. Duration is seldom more than a day or two†.

Muscles of Larynx. Those which open and close the glottis (1)

The posterior and lateral cricoarytenoids (2) The arytenoids. Those controlling the tension of the cords (1) The cricothyroids (2) The posterior cricoarytenoids (3) The thyroarytenoids (4) The vocales.

Those controlling the inlet of the larynx (1) The aryepiglottics (2) The thyroepiglottics.

Nerve supply of larynx is from the vagus. The superior laryngeal branch arises near the base of the skull and divides into the internal laryngeal nerve the sensory nerve of the larynx down to the level of the vocal cords and the external laryngeal nerve which supplies the cricothyroid muscle and the inferior constrictor of the pharynx the division taking place slightly below and anterior to the greater cornu of the hyoid bone. The external laryngeal nerve may be injured during the ligation of

hot) (3) Following stimulation of parasympathetic and depression of sympathetic nerves (4) Following irritation of mucosa of tracheobronchial tree (5) Following morphine (6) Following paraldehyde (slight) (7) Following barbiturates and cyclopropane (8) Due to histamine (note possibility of histamine release when curare is used) The pulmonary arteries like the arteries of the heart and the brain are more under the influence of circulating metabolites than under neurogenic influence. Inadequate suppression of reflex response because anaesthesia is too light is a potent cause of bronchospasm.

Cyclopropane constricts reflexly but locally depresses bronchial muscle. Vinylene and ethyl chloride first constrict but later by sympathetic stimulation dilate bronchi. Strong ether and chloroform vapour given suddenly constrict but if concentration is increased gradually both cause dilatation because of sympathetic stimulation and muscular depression.

**Collateral Respiration.\***—Collateral respiration is maintained through the pores of Kohn the so-called pulmonary alveolar vents. Alveoli in a lobule whose bronchiole is blocked by a mucous plug may remain aerated by diffusion of air through these pores from adjacent lobules. Release of histamine into blood-stream may cause oedema of the vents thus preventing collateral respiration. Pre-operative dosage with antihistamine drugs e.g. benadryl or pyribenzamine by mouth or of promethazine with or without pethidine intramuscularly may do good and may also reduce bronchiolar spasm. Hypoxia is said to cause oedema of the pores of Kohn.

Foreign material mucus blood etc. is removed from the bronchial tree by (1) Cilia (2) Peristaltic bronchiolar contractions (3) Cough reflex.

Cough reflex is most active in the region of the carina. A short sharp inspiration is followed by an explosive expiration against a closed glottis which suddenly opens allowing the built up pressure to be released and with it carrying upwards the foreign material. Spasm of whole bronchial tree often accompanies spasm of larynx. Bronchi are very irritable in pulmonary tuberculosis relatively insensitive in bronchiectasis.

**Movements of Bronchial Tree during Respiration**—During inspiration the bronchial tree elongates while the smaller bronchi and bronchioles dilate during inspiration and contract during expiration. These movements are passive unlike the peristaltic movement which helps to remove foreign bodies.

**International Nomenclature accepted by the Thoracic Society †—**  
**THE RIGHT LUNG—**

The upper lobe bronchus (no longer called eparterial)

**A THE RIGHT UPPER LOBE—**(1) The apical bronchus and segment (2) The posterior bronchus and segment (3) The anterior bronchus and segment

See Alley R. D. and Lindskog G. E. (1948) Pharmacological Factors Influencing Collateral Respiration. *Ann Surg* 1948 128 497  
 † *Thorax* 1950 5 3 222

**Bronchial Tree**—This subdivides progressively the terminal bronchioles being the last twigs. Air is carried by these twigs to the leaves of the tree where active interchange of gases is carried on. The respiratory unit is composed of respiratory bronchioles, alveolar ducts and sacs and pulmonary alveoli. These together form a primary lobule. Air in the alveoli is separated from blood in the capillaries by two thin layers of cells, the capillary and alveolar walls. Estimated area of respiratory epithelium—55 sq metres or over twenty five times the skin area.

Elastic tissue is plentiful right down to the alveoli. Recoil of the lung during expiration is probably due to this tissue.

Muscular fibres surround the air-ducts stopping at the end of the respiratory bronchioles. When strongly contracted they have a sphincter like action. They also produce a peristaltic movement to remove irritating foreign matter. This peristaltic action is depressed by morphine.

Bronchial arteries from the thoracic aorta supply as far as the end of the respiratory bronchioles. Distal to this blood-supply is from the pulmonary artery.

**Nerve supply**—

Each vagus passes to the back of the hilum and is joined by branches of the sympathetic from the second to the fourth or fifth thoracic sympathetic ganglia forming the anterior and posterior pulmonary plexuses. From there fibres go to the main bronchi and the pulmonary artery and their branches.

Motor nerves are from the vagus (constriction) and the sympathetic (dilatation). Afferent impulses pass along the vagus.

**Lining mucosa** is of ciliated epithelium. The cilia have a wave like motion resembling a cornfield in a breeze. The direction is upwards towards the mouth. The respiratory bronchioles are devoid of cilia but above this level they are plentiful and act most efficiently. They are not under nervous control. Action depressed by general anaesthetics, sedatives and cold air. Warm air stimulates ciliary action.

**Dilatation of Bronchi**—Is seen (1) On inspiration (together with lengthening) (2) On inhalation of cold air or gas (3) On inhalation of oxygen rich atmospheres (4) Following hypocapnia (5) Due to stimulation of the sympathetic or depression of the parasympathetic nerves (6) From depressant action of anaesthetic drugs on bronchial musculature e.g. ether (7) After local analgesics sprayed into bronchi and trachea (8) After atropine and scopolamine (9) After pethidine (10) After aminophylline (11) After bromethol (12) After adrenaline (13) After procaine (intravenous) (14) After phenothiazine derivatives (15) After mid or high extradural or subarachnoid block.

**Constriction of Bronchi**—Is seen (1) On expiration (together with shortening)—this narrowing causes more alveolar air to be expelled during expiration than is re-inhaled during inspiration (2) Following inhalation of hot gas or air (when a to and fro CO<sub>2</sub> absorber is used the inhaled atmosphere can become quite

hot) (3) Following stimulation of parasympathetic and depression of sympathetic nerves (4) Following irritation of mucosa of tracheobronchial tree (5) Following morphine (6) Following paraldehyde (slight) (7) Following barbiturates and cyclopropane (8) Due to histamine (note possibility of histamine release when curare is used) The pulmonary arteries like the arteries of the heart and the brain are more under the influence of circulating metabolites than under neurogenic influence. Inadequate suppression of reflex response because anaesthesia is too light is a potent cause of bronchospasm.

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range (from  $-40$  mm Hg to  $+40$  mm Hg) so that blood will be compressed from the auricles and great veins into the venous system producing venous oozing—true engorgement with blue venous blood. Cyanosis unassociated with respiratory obstruction is not accompanied by increased venous engorgement.

**Movements of Respiration**—The lungs are passive air is moved as by a bellows the walls of the thoracic cage causing the lungs to expand. During a normal inspiration the chest capacity is increased by a combination of descent of the diaphragm and spreading of the ribs. The negative pressure in the intrapleural space is made greater and is responsible for expanding the underlying lungs.

During inspiration all thoracic movements are increased although not equally. The diaphragm and the anterior parts of the ribs and sternum have the greatest movement so the parts of the lungs in contact with these areas expand the most. During inspiration the lung roots move downwards forwards and laterally while the trachea is stretched.

Anteroposterior diameter of the thorax is increased by the sternum and upper ribs moving upwards and forwards by the action of the external intercostals. The first and second ribs are immobilized by their attached scalene muscles and so become fixed points.

Transverse diameter is increased by the widening of the subcostal angle by the diaphragm and lower ribs.

Length of the thoracic cavity is increased by the diaphragmatic descent which pushes down the abdominal viscera. In light anaesthesia this pushing down may cause remarks from the surgeon.

**Expiration** is chiefly passive except when forced. The expanded thorax tends to resume its former position the elastic lungs recoil while the diaphragm is drawn up by the negative pressure in the thorax. The elastic recoil of lung tissue equals 5 mm Hg on expiration and 10 mm Hg on inspiration. In forced expiration the abdominal muscles contract and so help to push up the diaphragm while the internal intercostal muscles help to depress the ribs.

A *tracheal tug* synchronous with inspiration is often seen when the intercostal muscles are paralysed either by deep anaesthesia or by a muscle relaxant. T. A. B. Harris\* explains this as being due to the forcible action of the fibres of the crura of the diaphragm transmitted to the central tendon the pericardium the lung roots and the trachea in the presence of an atonic condition of the sternocostal fibres of the diaphragm.

**Muscles of Respiration.**—

**Inspiration** The diaphragm the external intercostals the scaleni (fixing ribs 1 and 2) the quadratus lumborum (fixing ribs 11 and 12).

\* *The Mode of Action of Anaesthetics* 1931 p. 122. Edinburgh E. & S. Livingstone Ltd.

International Nomenclature *continued*

- B THE MIDDLE LOBE —(4) The lateral bronchus and segment  
 (5) The medial bronchus and segment  
 C THE LOWER LOBE —(6) The apical bronchus and segment  
 (7) The medial basal (cardiac) bronchus and segment (8)  
 The anterior basal bronchus and segment (9) The lateral  
 basal bronchus and segment (10) The posterior basal  
 bronchus and segment

## THE LEFT LUNG —

- A THE LEFT UPPER LOBE —Upper division bronchus —(1) The  
 apical bronchus and segment (2) The posterior bronchus  
 and segment (3) The anterior bronchus and segment The  
 lingula (lower division) bronchus (4) The superior bronchus  
 and segment (5) The inferior bronchus and segment  
 B THE LEFT LOWER LOBE —(6) The apical bronchus and  
 segment (8) The anterior basal bronchus and segment  
 (9) The lateral basal bronchus and segment (10) The posterior  
 basal bronchus and segment

The absence of a medial basal (cardiac) segment involves  
 omission of segment (7) in left lung

**Lungs** —Each lung is invaginated from the hilum into the closed  
 sac of the pleura

The pleura consists of a pulmonary part a parietal part and a  
 diaphragmatic part The two layers are in contact gliding  
 one over the other the potential space being at a negative  
 pressure of about 5 mm Hg below atmospheric pressure The  
 elastic lungs collapse if air or liquid is admitted to the pleural  
 cavity If the thorax is laid open on both sides a pressure  
 of 7 mm Hg in the trachea is necessary to keep the lungs  
 from collapsing The right lung has three lobes the left lung  
 two

The lung roots are opposite the bodies of the 5th 6th and  
 7th dorsal vertebræ and each contains the bronchus pul-  
 monary artery two pulmonary veins bronchial vessels lymph  
 glands etc

When the pulmonary arteries are occluded nutrition of lungs is  
 partially carried out by bronchial arteries at normal systolic  
 pressure (pulmonary B P being lower than general systemic  
 B P) By extensive production of collateral bronchial arteries  
 respiratory function too can be partly taken over if pulmonary  
 vessels become acutely or chronically occluded

Expansion of the lungs at birth is due to descent of the diaphragm  
 which enlarges the thoracic cavity The lung follows the  
 thoracic walls while it becomes filled with air at atmospheric  
 pressure The lung is thus stretched and remains so throughout  
 life The pressure on the pleural surface is less than the pressure  
 on the alveolar surface

[Respiratory movements occur *in utero* and are easily depressed by  
 sedatives anæsthetics and carbon dioxide deficit

If the free flow of air into and out of the lungs is obstructed the  
 intrapulmonary pressures will be increased beyond the normal

**OPENINGS**—There are three large and several smaller ones

- 1 The aortic opposite the 12th dorsal vertebra slightly to left of midline Transmits the aorta azygos vein and thoracic duct
- 2 The œsophageal opposite the 10th dorsal vertebra Transmits the œsophagus and the vagi Slightly to left of middle line The left vagus is anterior the right vagus posterior The portal and systemic venous systems communicate at this point
- 3 The vena caval opposite the 8th dorsal vertebra Transmits the inferior vena cava and branches of the right phrenic nerve Slightly to right of middle line  
The sympathetic trunk passes on each side behind the internal arcuate ligament  
The right crus transmits the greater lesser and least right splanchnic nerves  
The left crus transmits the greater lesser and least left splanchnic nerves and the hemiazygos vein  
The musculophrenic and the superior epigastric vessels pierce the diaphragm anteriorly

**EXTERNAL INTERCOSTALS**—These are eleven in number on each side and extend from the tubercles of the ribs behind to a point near the costochondral junction in front each is then continued as the anterior intercostal membrane Fibres run downwards and forwards to the rib below and are attached to its upper border

**INTERNAL INTERCOSTALS**—These are smaller than the external muscles and are eleven in number on each side

They extend from the sternum to the angles of the ribs and are then carried back to the vertebral column as the posterior intercostal membranes Each passes in a downward and backward direction from the inner surface of the rib above to the upper border of the rib below The intercostales interni are sometimes regarded as being parts of the internal intercostal muscles

The two groups are supplied by the intercostal nerves and are believed to elevate the ribs and to prevent either indrawing or bulging during deep breathing

**INTRAPLEURAL PRESSURE**—

During inspiration 4.5 to 8 mm Hg below atmospheric pressure

During expiration 2.5 to 4 mm Hg below atmospheric pressure

**INTRAPULMONARY PRESSURE**—

During inspiration 2 to 4 mm Hg below atmospheric pressure

During expiration 1 to 4 mm Hg above atmospheric pressure

Primrose (*Brit J Anaesth* 1952 24 Jan 1) gives good reasons for believing that the intercostals are of no great importance in regard to movement of the ribs but serve to regulate tension in the intercostal spaces preventing retraction during inspiration and ballooning during expiration A rhythmic group of impulses goes to the external intercostals during inspiration and to the internal intercostals during expiration (Bronk and Ferguson)



**Muscles of Respiration** *continued*

Accessory muscles used in forced inspiration trapezius serratus anterior latissimus dorsi pectorales sternomastoid—they all raise the upper part of the thoracic cage

*Expiration* In normal breathing this is passive In forced breathing the muscles of the anterolateral abdominal wall and the transversus thoracis

**THE DIAPHRAGM**—A dome shaped partition between the thorax and the abdomen The peripheral part is muscular while the central tendinous part forms the muscles insertion It is responsible for 60 per cent of the air breathed during deep inspiration

Sternal part arises from the back of the xiphisternum the costal part from the inner surfaces of the lower six ribs and their cartilages interdigitating with the transversus abdominis

Vertebral part arises from the medial and lateral arcuate ligaments and median arcuate ligament connecting the crura

Medial arcuate ligament (lumbocostal arch) covers the psoas major and extends from the body of the 1st or 2nd lumbar vertebra to the transverse process of the 1st lumbar vertebra

Lateral arcuate ligament arches over the quadratus lumborum and extends from the tip of the transverse process of the 1st lumbar vertebra to the lower margin of the 12th rib

The crura blend with the medial arcuate ligaments the right arising from the anterior aspect of the bodies of the first three lumbar vertebrae the left from the first two lumbar vertebrae Fibres arching over the aorta from the crura form the median arcuate ligament

The central tendon trefoil in shape receives the muscle fibres from these various origins It is adherent to the pericardium above

**NERVE SUPPLY**—The phrenic and the lower six intercostal nerves

The phrenic nerve comes from C 4 with twigs from C 3 and C 5 It descends to the root of the neck lying anterior to the scalenus anticus which it crosses from lateral to medial

The right nerve on leaving the medial side of the scalenus anticus passes between the cervical pleura behind and the innominate vein

The left nerve leaves the scalenus anterior higher up crosses the first part of the subclavian artery and comes to lie between the pleura and the subclavian and the innominate vein

The nerve then on each side descends in the thorax towards the diaphragm along the medial surface of the pleura in front of the root of the lung

The phrenic nerve contains sensory and motor fibres the former supplying the pleura pericardium and diaphragm

Upper surface is related to the pericardium in the middle and the right and left pleura on each side

Under surface is related to peritoneum liver right and left kidneys and suprarenal glands the stomach and the spleen The pancreas kidneys and suprarenals lie on the crura

constant whereas the volume of tidal air varies with the depth of breathing. One normal inspiration produces more interchange of gases than occurs in several shallow breaths (as in depressed states or anaesthetic overdosage). The nearer the tidal volume is to the dead space volume the less efficient is the real respiratory exchange. Normally the alteration in the alveolar air produced by breathing just compensates for the interchanges taking place between the alveolar air and the blood of the pulmonary capillaries.

Expired air is a mixture of alveolar air and dead space air.

The air in the lower part of the trachea at the end of expiration is for all practical purposes identical with the alveolar air.

The dead space volume of the anaesthetic apparatus is the volume which is filled with air at the end of expiration; it is rebreathed by the patient during the next inspiration. The effective tidal volume is the tidal air minus the dead space volume.

For more complete account of terminology and symbols used in respiratory physiology see article by E. J. Moran Campbell 1957 *Brit J Anaes* 29 534.

**Vital Capacity**—The vital capacity and its subdivisions (inspiratory and expiratory reserve volumes and tidal volume) can be measured directly but may vary as much as 20 per cent from the expected norm. Decrease may result from—

1. Reduction in functioning lung tissue such as mass lesions of a lung, occlusion of a major bronchus, bronchial obstruction, pulmonary oedema, pneumonia, atelectasis, pulmonary fibrosis, pulmonary congestion and surgical excision of lung tissue.
- Inability to expand the lungs or thorax or move the diaphragm, limitation of chest expansion, e.g. lateral and prone positions, tight strapping, scleroderma, kyphoscoliosis, pain from operation wounds, fractured ribs, poliomyelitis, neuritis, etc. and myasthenia.

3. Limitation of descent of diaphragm, e.g. in pregnancy, ascites, abdominal tumours, pneumoperitoneum and phrenic nerve palsy.

4. Limitation of lung expansion, e.g. in pleural effusion, pneumothorax, diaphragmatic hernia, marked cardiac enlargement or pericardial effusion.

The vital capacity is increased after induced hypotension\* achieved by any means and is not due to vagal (ganglionic) block. The worse the pre-operative symptoms, the greater the benefit of induced hypotension. The mechanism may be pulmonary vascular depletion making more room for air in the alveoli. In addition, baroreceptor reflexes from the aortico-carotid sinuses may cause bronchodilatation during hypotension. The vital capacity may be reduced by 50–75 per cent in the first day or two after an upper abdominal operation—45–50 per cent after a lower laparotomy. This is chiefly due to pain.

**Muscles of Respiration continued**

**DIFFUSION RESPIRATION**—Adequate oxygenation can be maintained by insufflation of a large volume of oxygen through a fine catheter (to allow for the blow off of surplus oxygen) in the complete absence of respiratory movements.  $\text{CO}_2$  however accumulates but does not reach a high tension in the alveoli for 30–40 minutes

**COMPLIANCE**—The volume change per cm water pressure (litres per cm  $\text{H}_2\text{O}$ ). The lower the compliance the stiffer the lung and vice versa. Can be separated into lung compliance and chest wall compliance. It denotes the elastic properties of the lungs and thoracic cage

**MEAN MECHANICAL RESISTANCE**—The amount of pressure necessary to obtain a certain flow rate. Expressed as cm of water per litre per second. In emphysema there is high resistance and low compliance

**Lung Volumes —**

- 1 The tidal volume or depth of breathing is the volume of gas inspired or expired during each respiratory cycle. 500 ml is average volume
- 2 The inspiratory reserve volume (formerly complementary air minus tidal volume) is the maximal amount of gas that can be inspired from the end inspiratory position
- 3 The expiratory reserve volume (formerly reserve or supplemental air) is the maximal volume of gas that can be expired from the end expiratory level. 1200 ml is average volume
- 4 The residual volume (formerly residual air or residual capacity) is the volume of gas remaining in the lungs at the end of a maximal expiration. 1200 ml is average volume

**Lung Capacities —**

- 1 Total lung capacity (formerly total lung volume) is the amount of gas contained in the lung at the end of a maximal inspiration. 6000 ml is average capacity
- 2 Vital capacity is the maximal volume of gas that can be expelled from the lungs by forceful effort following a maximal inspiration. 4800 ml is average capacity
- 3 Inspiratory capacity (formerly complementary air) is the maximal volume of gas that can be inspired from the resting expiratory level. 3600 ml is average capacity
- 4 Functional residual capacity (formerly functional residual air) is the volume of gas remaining in the lungs at the resting expiratory level. 2400 ml is average capacity

**Volume of Air in the Lungs —**

**TIDAL AIR**—400–500 ml. The volume of air breathed in and out of lungs during quiet respiration. Represents about one eighth of that breathed during deep respiration. The air contained in the upper air passages is the dead space air and represents about 150 ml i.e. about 20–30 per cent of tidal air. To this may be added the dead space of the face mask breathing tubes etc. It takes no part in the actual respiratory exchange and remains

If a liquid is exposed to a gas at a given pressure and equilibrium is established the pressure of the gas in the liquid will equal the pressure of the gas in contact with it

- 4 HENRY'S LAW OF SOLUTION OF GASES (1803) — With temperature remaining constant the volume of gas going into solution in a given liquid is proportional to the partial pressure of the gas
- 5 GRAHAM'S LAW OF DIFFUSION OF GASES (1811) — The rate of diffusion of a gas varies inversely as the square root of its density
- 6 AVOGADRO'S LAW (1811) — Equal volumes of a gas under standard conditions of temperature and pressure contain equal numbers of molecules

CRITICAL PRESSURE — Pressure required to liquefy a gas at its critical temperature

CRITICAL TEMPERATURE — Temperature to which a gas must be cooled before it can be liquefied by pressure

VAPOUR PRESSURE — Pressure exerted by molecules of a gas escaping from a liquid. When vapour pressure equals atmospheric pressure liquid is at boiling point

SPECIFIC GRAVITY OF A GAS — This is the ratio of the weight of a unit volume to a similar volume of air (regarded as 1) under the same conditions of temperature and pressure

Specific gravities of gases used by anaesthetists

Trilene vapour	4.35	Chloroform vapour	4.12	Ethyl chloride	
2.28	Ether vapour	2.6	Vinesthene vapour	2.2	Nitrous oxide
1.53	Carbon dioxide	1.5	Cyclopropane	1.46	Oxygen
1.1	Air	1	Ethylene	0.97	Nitrogen
0.6	Helium	0.13			

SOLUBILITY OF GASES IN VOLUMES PER CENT AT 38°C AND 760 MM Hg PRESSURE —

	Water	Plasma	Blood
Oxygen	2.37	2.3	2.3
Nitrogen	1.2	1.2	1.1
Carbon dioxide	55.5	54.1	54.1

Air at atmospheric pressure or tension exerts a pressure of 760 mm Hg. At higher altitudes or at lower barometric pressures percentage composition of air does not alter but partial pressure of each gas is reduced. Barometric pressure is reduced 20 mm Hg for each 1000 ft increase in altitude.

The following table shows the oxygen and carbon dioxide tensions —

	Oxygen Tension mm Hg	Carbon Dioxide Tension mm Hg
Alveolar air	100	40
Mixed venous blood	40	46
Arterial blood	80-90	40

The  $\text{CO}_2$  tension in the alveoli is  $40 \pm 5$  mm Hg. The oxygen tension is  $100 \pm 5$  mm Hg.

## Vital Capacity continued

**DIFFUSION**—This can now be measured and in the lungs may be impaired as a secondary factor in disease or it may occur specifically as alveolar capillary block which has been found present in some cases of Bocck's sarcoid of the lung beryllium granulomatosis asbestosis pulmonary scleroderma alveolar cell carcinoma sulphur dioxide poisoning and certain metastatic lung lesions

**ALVEOLAR AIR**—The air in contact with the pulmonary capillaries which therefore carries out gaseous interchange with the blood

Made up of the expiratory reserve volume and the inspiratory reserve volume—about 2500 ml

**MINUTE VOLUME**—The volume of air breathed each minute Equals the tidal air multiplied by the number of respirations per minute The normal pulmonary ventilation is 5 to 8 litres per minute If the minute volume is reduced care must be taken to prevent oxygen lack and carbon dioxide accumulation by assisting the minute volume by intermittent positive pressure High extradural or spinal analgesia cyclopropane thiopentone or deep ether may cause a reduced minute volume and so may muscle relaxants A minute volume which is reduced may be adequate for the patient's oxygenation but inadequate to remove his carbon dioxide

**AVERAGE COMPOSITION OF AIR—**

	Inspired Air	Expired Air	Alveolar Air
Oxygen	21	16.3	14.2
Carbon dioxide	0.04	4	5.5
Nitrogen	79	80	80

Atmospheric air contains less than 1 per cent of water vapour

The lungs contain over 6 per cent of water vapour

**Laws of Gases**—The kinetic theory of gases postulates that the molecules of a gas in an enclosed space are constantly moving and so are constantly bumping into each other and into the walls of the enclosing space These bumpings give rise to the pressure or tension of the gas in the space The greater the number of molecules the greater will be the tension

Temperature increases movement and so increases tension

1. **BOYLE'S LAW (1662)**—The volume of a gas varies inversely with the pressure it is subjected to the temperature remaining constant
2. **CHARLES'S LAW (1787)**—At constant pressure the volume of a gas is proportionate to its absolute temperature
3. **DALTON'S LAW OF PARTIAL PRESSURE**—The pressure of a gas in a physical mixture of gases equals the pressure which that quantity of gas would produce were it alone Thus the total pressure of a mixture of gases equals the sum of the partial pressures of the individual gases The partial pressure of a gas in a mixture is proportional to its percentage by volume in the mixture

In arterial blood 40-55 volumes per cent of carbon dioxide are carried the greater part as bicarbonate

1 g of hæmoglobin carries 1.34 ml of oxygen when fully saturated

15 g of hæmoglobin (the normal amount in 100 ml of blood) carries 20 ml oxygen—actually about 19 ml of oxygen saturation not being maximal. The tissues require about 250 ml of oxygen each minute—the basal oxygen

**Oxygen Carriage in Blood**—The dissociation curve of hæmoglobin worked out for temperature of 37° C and CO<sub>2</sub> tension of 40 mm Hg shows the relation between the partial pressure of oxygen (abscissæ) and the percentage saturation of the hæmoglobin (ordinates). It shows that at 100 mm Hg partial pressure hæmoglobin is 95 per cent saturated whereas at 70 mm Hg hæmoglobin saturation is still 90 per cent. The dissociation curve may be altered by changes in the pH. Carbon dioxide increase causes a flattening of the curve to the right. An increase in temperature has a similar effect. Normally arterial blood is at a tension of 100 mm Hg at which hæmoglobin is 95 per cent saturated and the oxygen content is 19.5 volumes per cent. 0.3 ml of this being dissolved in plasma. Venous blood has an oxygen tension of 40 mm Hg and its oxygen content is 14 volumes per cent.

**Results of Inhalation of Pure Oxygen**—If 100 per cent oxygen is inhaled the tension in arterial blood increases to 700 mm Hg and hæmoglobin is fully saturated. The arterial oxygen content increases from 19.5 volumes per cent to 23.5 volumes per cent and of this increase 0.5 ml is due to increased combination with hæmoglobin the remainder to increased solution in plasma. As no more oxygen is used by the body the venous blood also contains more oxygen. The saturation of hæmoglobin in mixed venous blood is 80 per cent and the partial pressure of oxygen is 60 mm Hg so after breathing pure oxygen there is a 50 per cent increase in partial pressure of oxygen in venous blood over normal.

The oxygen and carbon dioxide dissociation curves in blood stored in an acid-citrate dextrose medium are shifted to the left and oxygen and carbon dioxide volumes are reduced. Such changes are progressive with storage and the effect lasts several hours after transfusion. As a result of these shifts the blood of an anæmic patient may for a few hours after transfusion be unable to release as much oxygen as it did before.\*

Bohr has shown that when the reaction of blood becomes more acid as with the addition of carbon dioxide or lactic acid oxygen hæmoglobin parts with its oxygen more readily. This is an argument but not a convincing one for partial rebreathing in anaesthesia when the blood carbon dioxide level is raised. In practice there is no evidence that a low blood CO<sub>2</sub> level interferes with tissue oxidation.

**Exchange of Gases in Lungs**—Diffusion is aided by differences in pressure or tension. As inspired air is sucked into the alveoli it mixes with air already there and so its oxygen becomes diluted its carbon dioxide increased and water vapour added. It is the partial pressure of the oxygen (101.2 mm Hg) which drives it into the blood across the pulmonary epithelium and the walls of the pulmonary capillaries as the pressure of oxygen in these capillaries is about 40 mm Hg. The result is that oxygen tension in the capillaries rises almost to the same level as it is in the alveolar air so that the blood leaving the lungs in the pulmonary veins approximates that of the alveolar air in its oxygen tension.

The following table shows the partial pressures of gases in the lungs and blood—

	INSP. AIR	EXPIRED AIR	ALVEOLAR AIR	ARTERIAL BLOOD	VENOUS BLOOD
<b>Oxygen</b>					
Tension in mm. Hg	13.35	11.6	101.2	100.0	40.0
Volumes per cent	20.9	16.0	15.0	19.0	16.0
<b>Carbon Dioxide</b>					
Tension in mm. Hg	0.3	28.5	40.0	40.0	46.0
Volumes per cent	0.04	4.5	5.6	5.0	5.5
<b>Water Vapour</b>					
Tension in mm. Hg	596.45	576.0	576.0	570.0	500.0
Volumes per cent	79.0	94.0	79.0	83.0	83.0
<b>Water Vapour</b>					
Tension in mm. Hg	3.0	47.0	47.0		
Volumes per cent	0.00	0.5	0.5		

**Exchange of Gases in Blood**—Again gas passes from a zone of high to a zone of low pressure or in other words the pressure gradients enable oxygen to diffuse inwards from alveolar air to capillary blood and carbon dioxide to diffuse in the reverse direction. The diffusion coefficient of carbon dioxide (500) is greater than that of oxygen (25-45) i.e. it diffuses twenty times as readily so this compensates for the lower pressure gradient of this gas as compared with that of oxygen.

This slower diffusion of carbon dioxide than oxygen explains why a tidal exchange sufficient to keep the arterial oxygen level up to normal may be too small to remove adequately the carbon dioxide. This is not inconsistent with the fact that carbon dioxide passes more rapidly across the alveolar membrane than oxygen because of its greater solubility in water.

In whole blood 0.24-0.3 volumes per cent of oxygen are carried in simple solution.

In whole blood 2.5 volumes per cent of carbon dioxide are carried in simple solution.

In arterial blood 10-20 volumes per cent of oxygen are carried the greater part combined with hemoglobin.

an enzyme to form carbonic acid  $\text{H}_2\text{CO}_3$ . (This enzyme likewise accelerates the splitting up again into water and carbon dioxide it is not present in plasma only in the red cells)  $\text{H}_2\text{CO}_3$  being a stronger acid than reduced hæmoglobin attracts base from the latter in the form of potassium (hæmoglobin exists chiefly as potassium hæmoglobinate) Thus  $\text{KHCO}_3$  is formed. Bicarbonate ions now leave the cells while chlorine ions from the plasma take their place. Thus plasma chloride decreases while cell chloride and plasma bicarbonate increase. This is the so-called *chloride shift* or Hamburger phenomenon. The newly formed bicarbonate arriving in the plasma from the cells combines with the sodium there to form sodium bicarbonate. This sodium is set free when sodium chloride is split to enable the chloride ion to shift into the red cells there to combine with the potassium ions. The purpose of the chloride shift is to allow some of the carbon dioxide entering the red cells to be converted into bicarbonate and in this form to be carried in the plasma.

Reverse changes occur in the lungs the shift working the other way. Reduced hæmoglobin becomes oxyhæmoglobin and carbon dioxide is excreted into the plasma and so diffuses into the alveolar air. Reduced hæmoglobin being a weaker acid takes up carbon dioxide in the tissues more readily than oxyhæmoglobin. But oxyhæmoglobin when formed in the lungs liberates carbon dioxide more easily. In the lungs the relatively more alkaline reduced hæmoglobin unites with oxygen and so becomes relatively acid and thereby attracts the potassium ion from the potassium chloride so liberating a chloride ion which diffuses out of the cells while bicarbonate ions diffuse in. As this occurs carbon dioxide is liberated and diffuses into the plasma and alveolar air.

In addition to transporting  $\text{CO}_2$  sodium bicarbonate also acts as the alkali reserve neutralizing strong acids which may enter the blood stream. The pH of plasma depends on ratio of  $\text{H}_2\text{CO}_3$  to bicarbonate. Breathing thus assists in the pH control of the blood.

Acidosis is metabolic if plasma bicarbonate is reduced and respiratory if the blood carbon dioxide is increased the plasma bicarbonate remaining more or less normal.

**Regulation of Respiration** — Rhythmical breathing is controlled by cells in the respiratory centre. It has its own rhythm and is influenced by peripheral stimuli and humoral states especially the tension of carbon dioxide. Periodic outbursts of inspiratory impulses originate from the centre in the middle third of the medulla. While essentially automatic the centre may be influenced from many sources. (1) From changes in oxygen and carbon dioxide tension in the blood. (2) By changes in body temperature. (3) By Hering Breuer reflexes. (4) By carotid body reflexes largely oxygen lack. (5) By proprioceptive impulses from intrathoracic structures and from muscles and joints throughout the body so that respiration can fit in with exercise and movement, etc. (6) By reflexes which modify rhythm during



**Passage of Oxygen from Blood to Tissues**—Oxygen passes from alveolar air to blood by diffusion. The saturation of hæmoglobin depends on the oxygen tension of the blood or the amount of oxygen in solution in the plasma. This latter depends on the oxygen pressure in the alveoli.

Arterial blood containing a high oxygen content in solution with oxyhæmoglobin in equilibrium with it comes into contact through the capillary epithelium in the tissues with fluid poor in oxygen. This results in oxygen flowing across the capillary membranes so that arterial plasma and oxyhæmoglobin cease to be in equilibrium. Oxygen thus passes from the oxyhæmoglobin to the plasma. The difference in partial pressure of oxygen between red cell and tissue cell causes a steady flow of oxygen from the red cells to the tissue cells.

**Carriage of Carbon Dioxide in Blood**—Total amount of carbon dioxide carried in blood is about 50-60 volumes per cent. Venous blood contains 5-10 per cent more carbon dioxide than arterial blood, its carbon dioxide combining power being greater.

The tension of carbon dioxide in arterial blood and alveolar air is 40 mm Hg. In venous blood at rest it is 46 mm Hg. Carbon dioxide output about 200 ml per minute.

Carbon dioxide is carried in blood in three forms—

- 1 As dissolved carbon dioxide in plasma—from 3 per cent to 5 per cent. The amount of pre-dissolved carbon dioxide determines the partial pressure of the gas and it is this difference in partial pressure of carbon dioxide which accounts for carbon dioxide transport. The partial pressure of carbon dioxide in the body cell is greater than that in the capillary blood so the gas passes into the blood. The partial pressure of carbon dioxide in mixed venous blood is 46 mm Hg and is greater than that in the alveoli which is 40 mm Hg, so the gas passes from the blood to the alveoli and is exhaled.
- 2 As carbamino compounds formed by the combination of carbon dioxide with  $\text{NH}_2$  groups from hæmoglobin in the red cells—called carbamino hæmoglobin or carbhæmoglobin. Carbon dioxide also combines on a small scale with plasma proteins. Reduced hæmoglobin can take up more carbon dioxide in this way than oxyhæmoglobin. The formation and dissociation of carbamino compounds is a rapid process. About 2 per cent to 10 per cent of the total blood carbon dioxide is carried in this way, the amount depending on the degree of oxygenation of hæmoglobin.
- 3 As bicarbonate chiefly of sodium and potassium. Mainly in plasma but also in the corpuscles. Whenever carbon dioxide enters or leaves the blood stream an interchange of ions takes place across the membranes of the red cells which have the property of admitting negatively charged anions such as bicarbonate and chloride ions while holding back positively charged cations such as sodium and potassium ions. About 80-90 per cent carried in this way.

When in the tissue capillaries carbon dioxide enters the red cells it rapidly combines with water with the help of carbonic anhydrase.

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**Regulation of Respiration continued**

talking swallowing etc (7) Reflexes from the hypothalamus and cerebral cortex. The respiratory centre is in the pons and upper medulla (Legallois 1812). It consists of (1) An inspiratory centre (2) An expiratory centre and (3) A pneumotaxic centre which periodically inhibits the constant stream of impulses from the inspiratory centre converting them into alternate inspiration and expiration. It is connected with the phrenic and intercostal nerves by fibres running in the anterior and lateral columns of the white matter of the cord and on the afferent side with the vagus sinus and aortic nerves. The centre is influenced by the will and by emotional states.

Hering Breuer reflexes have been known since 1868. Afferent impulses travel up the vagus and cause inspiration to be inhibited when alveoli are inflated; expiration is inhibited when the alveoli are deflated sufficiently. Stretch and deflator receptors are present in alveolar walls. Hypoxia exaggerates this reflex while hyperpnœa abolishes it. All volatile and gaseous anaesthetics increase excitability of the pulmonary receptors which are stimulated by inflation. Deflation receptors are first stimulated later depressed by chloroform and ethyl ether. Trichlorethylene sensitizes deflation receptors without subsequent depression—hence tachypnœa seen with this agent. Thiopentone depresses the respiratory centre but exaggerates the Hering Breuer reflex in direct proportion to depth of anaesthesia; thus apnœa will result following reservoir bag pressure under this type of anaesthesia sufficiently long for radiological investigation of the bile ducts for example.

Heat cold and pain stimulate breathing while impulses arise in skeletal muscles during active exercise and have the same effect. Traction reflexes arising in the abdomen and chest stimulate respiration. Breathing is inhibited during swallowing (glossopharyngeal nerve). It is also inhibited by local irritation of the larynx by such vapours as strong ether or by foreign bodies. Continuous stimulation of the inspiratory centre results in apneusis—maintenance of the inflated position.

**EFFECTS OF CHEMICAL AGENTS ON RESPIRATORY CENTRE**—Carbon dioxide influences the actual cells of the respiratory centre and a blood carbon dioxide tension increase of 5 mm Hg will stimulate respiration whereas the blood oxygen tension has to go down by about 60 mm Hg (from the normal 100 mm Hg to 40 mm Hg) before hypoxia stimulates the centre. This corresponds to breathing 12 per cent oxygen instead of the normal 21 per cent or breathing air at 13 000 ft. Thus we breathe primarily to get rid of carbon dioxide! Carbon dioxide is the normal and dominant respiratory stimulus. Ventilation which is adequate to get rid of carbon dioxide is more than adequate to replace oxygen in the alveolar air.

Oxygen lack depresses the respiratory centre. The oxygen tension is an important respiratory stimulant only when the patient is breathing hypoxic mixtures.



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If carbon dioxide is breathed the carbon dioxide in the alveolar air does not diffuse out so readily i.e. the gradient is not as steep. This results in a raised alveolar carbon dioxide tension with a similar change in the arterial blood. The latter causes a stimulation of the respiratory centre.

If more than 6 per cent of carbon dioxide is inspired no amount of hyperventilation can keep the alveolar air below that figure so the arterial carbon dioxide rises producing an acidæmia or respiratory acidosis. If alveolar carbon dioxide is kept lower than normal by forced breathing apnoea results.

**Acid-base Balance and Anæsthesia** \*—Changes occur in the pH of the blood during anæsthesia which are respiratory in origin. In the conscious state the pH is controlled very carefully by altering sensitivity of the respiratory centre but during anæsthesia with central and peripheral respiratory depressants this adjustment is lost. By using positive pressure artificial respiration a normal acid base balance can be maintained but this may require a minute volume double that in the resting conscious state. One reason for this great increase may be the alteration of the pulmonary hæmodynamics caused by the positive pressure used in ventilation. This state of affairs is again much worse in the prone lateral and Trendelenburg positions. Respiratory acidosis may cause a shock like state to develop encourages hiccup and increases the need for intravenous thiobarbiturates.

#### **Carotid and Aortic Reflexes and Respiration** —

*Pressoreceptors* are present in the carotid sinus a slight enlargement of the common carotid artery just before its bifurcation and the internal carotid artery. They are also present in the aortic arch. Stimulation of the nerve-endings (stretch receptors) in the outer wall of the sinus and aorta inhibits respiration (Heymans 1929). This occurs when pressure rises. Pressoreceptors are also present in the great veins right auricle and pulmonary artery and increased pressure here may stimulate respiration. Such reflexes may be involved in the dyspnoea of heart failure and pulmonary embolism.

There is evidence that pressoreceptors when stimulated by low blood pressure can produce reflex bronchial dilatation.

*Chemoreceptors* are present in the carotid body and the aortic bodies small masses of polyhedral cells forming a rich network of capillaries. The carotid body is in relationship to the origin of the occipital artery or the external carotid artery just after its commencement from the bifurcation of the common carotid artery. The aortic body lies between the descending aorta and the pulmonary artery. When stimulated by decreased oxygen tension or increased carbon dioxide tension there is increase in rate and depth of breathing. Stimulation begins when oxygen tension falls to 70 mm Hg at which point the arterial blood is 92 per cent saturated with oxygen i.e. about equal to that saturation resulting from inhalation of a mixture containing 18 per cent oxygen or by ascending to a height of 4000 ft.



**Carotid and Aortic Reflexes and Respiration continued**

Thus hypoxia directly depresses the respiratory centre but secondarily stimulates it via this reflex. Stimulation only occurs if plasma oxygen tension is reduced not blood-oxygen content (as in anaemia). Chemoreceptors are important when the respiratory centre is depressed as by barbiturates morphine etc. when the resulting hypoxia stimulates respiration reflexly. If at such times oxygen in excess is given the sole remaining stimulant to breathing is removed and apnoea may follow. With ether respiratory centre is not so greatly depressed.

**Carotid and Aortic Reflexes and Circulation —**

*Pressoreceptors* when stimulated by increase in pressure inside artery or from outside its walls cause bradycardia vasodilatation and a lowered blood pressure (reflex stimulation of vagal nucleus). When instead decrease of pressure occurs tachycardia vasoconstriction and raised blood pressure result (Hering 1927). There is also an increase in adrenaline secretion. The carotid sinus syndrome is seen in subjects with an irritable carotid sinus reflex mechanism. It comprises bradycardia and hypotension and is seen when the sinus is stimulated from without by pressure or electricity (e.g. during thyroidectomy).

*Chemoreceptors* when stimulated by oxygen lack or carbon dioxide excess produce vasoconstriction and rise in arterial blood pressure. The chemoreceptors are also stimulated by lobeline acetylcholine cyanide coramine and nicotine. Acetylcholine is probably the chemical mediator within the carotid body.

Afferent arc of these reflexes is via sinus nerve a branch of the glossopharyngeal nerve ascending between internal and external carotid arteries also the aortic nerve via the vagus which also has connexions with the superior cervical sympathetic ganglion.

Efferent arc is via vagus to heart and via sympathetic outflow to vasomotor mechanism.

**Hypercapnia** (υπέρ (hyper) over + καπνός (kapnos) smoke) — Excess of carbon dioxide in the blood. May cause fine tremors of arms and fingers and when severe convulsions. It depresses auriculo-ventricular conduction so that if  $pH$  is 7 complete heart block results. Weakening of beat and arrhythmia may occur if carbon dioxide excess is maintained. The rapid reversal of hypercapnia may cause cardiac arrhythmia or even ventricular fibrillation perhaps due to a rise in the level of blood potassium.

**Hyperventilation** — This causes a reduced carbon dioxide content in the plasma increased blood alkalinity resulting in increase of urinary secretion and urinary phosphate and urinary alkalinity. Increase in blood lactate appearance of acetone bodies in the urine. The skin becomes pale moist and cold owing to vasoconstriction—a peripheral effect. The cerebral blood flow is reduced. Clinical changes due to hyperventilation are seldom harmful. The pain threshold is raised and analgesia potentiated.

**L Hyoscyamine \***—The *l*-iso-isomer of tropine tropate (bellafoline) Atropine itself is racemic an equal mixture of *d* and *l* tropine tropate and is not found in the belladonna plant in nature but is formed during the process of isolation Hyoscyamine in doses of gr  $\frac{1}{8}$  is said to be a better drying agent than atropine It causes drowsiness and tachycardia and sometimes other undesirable side-effects

**Methantheline Bromide** (banthine) —Has been recommended as a drying agent before anaesthesia† (1 mg per year) and so has **oxyphenonium bromide†** (antrenyl)

#### Note on Innervation of Salivary Glands —

**PARASYMPATHETIC** —To sublingual and submaxillary glands Pre ganglionic fibres from cells in the superior salivary nucleus through facial lingual and chorda tympani nerves to glands directly Post ganglionic cells lie along the chorda tympani the submandibular ganglion These are secretory and vasodilator fibres

To parotid pre ganglionic fibres pass from cells in the inferior salivary nucleus via the glossopharyngeal nerve its tympanic branch (nerve of Jacobson) the lesser superficial petrosal nerve to the otic ganglion Post ganglionic fibres pass from the otic ganglion to the auriculotemporal branch of the fifth nerve Secretory and vasodilator fibres

**SYMPATHETIC** —From cells in the intermediolateral horn of the first and second thoracic segments via anterior roots of T 1–T 2 and corresponding white rami to the cervical sympathetic chain Post ganglionic fibres come from the superior cervical ganglion and go to the external carotid and internal maxillary arteries and so to glands These are vasoconstrictor fibres

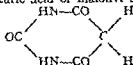
#### Advantages and Disadvantages of Atropine and Scopolamine — ADVANTAGES —

- 1 Inhibits secretion which might interfere with airway or be aspirated into chest This advantage is greater than the disadvantage of tenacious thick mucus formation
- 2 Lessens laryngeal spasm especially when intravenous barbiturates are used
- 3 Stimulates respiration depressed by opiates or pethidine
- 4 Scopolamine produces sedation and amnesia

#### DISADVANTAGES —

- 1 Interferes with pupil reactions
- 2 May cause thick tenacious mucus formation which is removed from the bronchial tree only with difficulty
- 3 May produce tachycardia
- 4 May owing to its anhidrotic effect be liable to cause a rise in temperature
- 5 Scopolamine may cause restlessness especially in the aged

**barbiturates** —The parent molecule from which the barbiturates are derived is barbituric acid or malonyl urea



This was described in 1881 by Conrad and Guthzeit and introduced into medicine by V. Mering and Emil Fischer. Four groups are described —

- 1 The ultra short acting e.g. thiopentone hexobarbitone kemithal mainly detoxicated in the liver
- 2 The short acting e.g. nembutal secobarbital sodium soneryl detoxicated in the liver
- 3 The medium acting e.g. sodium amytal dial ispral neonal phendorm partly eliminated unchanged partly detoxicated in the liver
- 4 The long acting e.g. barbitone phenobarbitone eliminated unchanged by the kidneys

The short acting barbiturates form useful pre anaesthetic sedatives. They produce sleep allay anxiety depress the central nervous system without producing the same degree of respiratory depression as does morphine. They probably depress the hypothalamus. As they are not analgesic they may give rise to restlessness and loss of self control in the presence of unrelieved pain. Beecher\* however thinks that pentobarbital does relieve pain by producing a pharmacological leucotomy a reversible depression of the interuncinal spread of pain impulses perhaps between the thalamus and the cortex. They reduce the B.M.R. and the blood pressure and they depress the renal and hepatic function. Their effect on the E.E.G. is similar to that of natural sleep. They traverse the placental barrier. They reduce the tone of the gut.

To treat overdosage Nilsson† advises as follows regarding the state of depression as prolonged anaesthesia. Avoidance of analeptics gastric lavage only if the pharyngeal reflexes are active leaving inside stomach 20 g of activated charcoal in water continuous endotracheal oxygen insufflation with intermittent tracheo-bronchial toilet and suction frequent movement of patient administration of sulphadiazine and penicillin careful biochemical control of blood aiming at slight alkalosis to barbiturate excretion injections of vitamin C to increase vascular permeability for threatened acute pulmonary oedema 50 per cent glucose intravenously for circulatory collapse blood or dextran for cardiac failure strophanthin 1-1½ intravenously to raise blood pressure quickly 25-50 mg intravenously Sleep may be reinduced by 5-10 mg and also by glucose given to recovering patients after barbiturate intoxication ‡

ganglia to the cardiac plexus which also receives post ganglionic fibres direct from the upper five or six thoracic sympathetic ganglia. The efferent fibres to the lungs come from T 2 to T 6 or T 7 and synapse in the stellate and upper thoracic ganglia from which fibres pass to the posterior pulmonary plexus. The œsophagus derives its nerve supply from T 4 to T 6.

*Visceral afferent fibres* carrying pain from the heart and aorta (T 1-T 5) accompany the cervical and thoracic cardiac nerves and enter the cord via the white rami of the upper five thoracic nerves to have their cell stations in the corresponding posterior root ganglia.

The thoracic part of the sympathetic trunk usually consists of ten or eleven ganglia and their connecting fibres which lie anterior to the heads of the ribs behind the pleura in the upper part but on the sides of the vertebral bodies in the lower part.

**ABDOMEN**—Pre ganglionic fibres from T 5 to L 2. These pass through the ganglia of the sympathetic chain to form the three splanchnic nerves. Fibres from L 1 and L 2 reach the aortic renal plexus directly. Synapse occurs in cœliac (solar) plexus from which post ganglionic fibres reach the viscera with the arteries.

The sympathetic system probably contains vasodilator fibres e.g. to blood vessels of muscle but vasodilatation is mainly humoral.

*Visceral afferent fibres* from the alimentary canal and its offshoots travel with the splanchnic nerves entering the cord on their way to the posterior root ganglia via the white rami from the fifth thoracic to the second lumbar nerves. The visceral afferent supply to the abdominal organs is as follows\*—The gall bladder T 4-T 10. The liver T 6-T 10. The pancreas T 6-T 10. The small intestine T 8-T 11. The cæcum and appendix T 10-T 12. The colon to the splenic flexure T 11-L 1. The splenic flexure to the rectum L 1-L 2. The suprarenals T 10-L 2. The kidneys T 11-L 2. The ureters L 1-L 2. The urinary bladder T 11-L 2. The uterus T 11-L 2.

Motor fibres to the alimentary canal travel with the splanchnic nerves.

Branches of the phrenic join the cœliac plexus (Hovelague 1927) as also do twigs from the vagus.

**MAINTENANCE OF BLOOD PRESSURE**—The blood pressure is the pressure of blood on the arterial walls the systolic blood pressure being the maximal pressure during propulsion of blood the diastolic pressure the minimal pressure occurring at the end of diastole. The difference between them is the pulse pressure. Depends on (1) Force of the heart (2) Peripheral resistance (3) Volume of blood (4) Viscosity of blood (5) Elasticity of arterial walls.

**Maintenance of Blood pressure continued**

After induction of general anaesthesia there is a rapid onset of vasodilatation due to (1) A direct depressive action of the agent on the vasomotor centre and (2) Direct action on the vessels. Later on vasoconstriction is seen especially in prolonged operations and is an attempt at circulatory re-adjustment to maintain blood pressure.

## NOTE ON AUTONOMIC\* NERVOUS SYSTEM

**Thoracico lumbar Outflow (Sympathetic†)**—This consists of a series of connector cells in the intermediolateral horn of grey matter together with their connector fibres which are B type fibres white and medullated. As they travel to peripheral ganglia they are called pre ganglionic. They synapse at these peripheral ganglia with excitor cells which in their turn give off fibres which are of course post ganglionic and usually are non medullated and grey in colour. These run to the viscera and are C type fibres. Both these are efferent.

The afferent neurones of the viscera are not strictly autonomic although their fibres travel with autonomic fibres. Each travels from end organs in the viscera has a cell station in a ganglion of the posterior root and sends a central process into the grey matter.

Anatomically the sympathetic outflow extends from the first thoracic to the second or third lumbar segments of the cord. In each segment the connector cells in the intermediolateral horn send off fibres which leave with the anterior nerve root. The fibres travel across the subarachnoid and extradural spaces form part of the mixed spinal nerve and the anterior primary ramus and then as *white rami* join the corresponding paravertebral ganglion of the sympathetic chain. Here it may end synapsing with an excitor cell and being continued as a post ganglionic fibre or it may pass uninterrupted through the ganglion to synapse with an excitor cell in a collateral ganglion (abdomen) or other paravertebral ganglion (e.g. superior cervical ganglion). In the thoracic region each white ramus measures about 1 cm in length this increases to 2-3 cm in the lumbar region.

**Craniosacral Outflow (Parasympathetic‡)**—Fibres leave in the 3rd 7th 9th and 10th cranial nerves. The 1st is important to the spinal anaesthetist as it is not blocked by spinal analgesia. Stimulation of the vagal nerve endings e.g. in the stomach during gastrectomy results in discomfort to a conscious patient an effect removed by the infiltration of a local analgesic into the para-oesophageal tissues near the cardia.

Sacral fibres leave the cord with the second third sometimes fourth sacral nerves. They leave in the cauda equina and after the anterior sacral rami have passed through the anterior sacral

\* Term first used by Langl y n 879

† Term first used by D nish n tomist Winslow n 73

‡ Term first used by British physiologist Langl y in 1905

foramina the white rami are given off. As pelvic nerves or *nervi erigentes* one on each side they do not pass through the sacral sympathetic chain but run direct to the hypogastric ganglia and thence to the walls of the pelvic viscera. Their post ganglionic or excitor fibres are given off from cells in the walls of the genitalia bladder and rectum. They cause contraction of the hollow viscera relaxation of sphincters and vasodilatation. In addition visceral afferents travel with these nerves.

**Ganglia —**

**LATERAL GANGLIA** —This is the sympathetic chain and is composed of three cervical eleven thoracic four lumbar and four sacral ganglia. The chain receives white rami from T 1 to L 3 while from it every one of the spinal nerves receives a grey ramus communicans which is an excitor or post ganglionic fibre. One sympathetic ganglion may give off several grey rami. The grey rami carry pilomotor and vasomotor impulses and secretory impulses to the sweat glands. They reach their destinations via spinal nerves (each nerve receives a grey ramus) and blood vessels. The inferior cervical and first thoracic ganglia unite to form the stellate ganglion at the level of the seventh cervical vertebra posterior to the subclavian artery. In the neck the chain of ganglia lies anterior to the transverse processes on the deep fascia covering the longus colli and longus capitis muscles. The thoracic ganglia lie anterior to the heads of the ribs the lumbar ganglia irregular in shape number and position lie in front of the anterolateral aspect of the bodies of the lumbar vertebrae. Certain pre ganglionic fibres (connector) do not relay in the sympathetic chain ganglia from the fifth thoracic to the second or third lumbar segment they pass directly as splanchnic nerves to the coeliac plexus where excitor cells are placed and from where post ganglionic fibres are distributed to the abdominal viscera. The coeliac plexus receives in addition to the sympathetic connector fibres fibres from the vagus and the phrenic. It is continued downwards in the retro peritoneal pre aortic region where it becomes the hypogastric plexus. Each pre ganglionic (connector) fibre synapses with over a score of excitor or post ganglionic neurones.

**COLLATERAL GANGLIA** —Example the coeliac

**TERMINAL GANGLIA** —Auerbach's (1857) and Meissner's (1864) plexuses

**Afferent Side of the Autonomic System** —There are afferent autonomic pathways carrying painful impulses from the viscera with cell stations in the posterior root ganglia of the spinal nerves. They are similar to somatic afferent fibres—are not divided into pre and post ganglionic neurones. The long peripheral fibres travel from plexuses in the viscera with sympathetic fibres through grey and white rami to posterior roots. While the majority of these afferent fibres are medullated and larger in size than the efferent fibres some are non medullated. All the fibres enter the cord between T 1 and L 3 except those from the bladder rectum prostate cervix uteri and lower colon which pass to the cord via *nervi erigentes*.

**Autonomic Nervous System continued**

**Physiology of Autonomic System** — The thoracico-lumbar outflow the sympathetic produces widespread diffuse effects. It activates the body for defence and is *katabolic*.

The craniosacral outflow the parasympathetic produces localized effects and is *anabolic*. When an organ is innervated by both systems they are antagonistic in effect.

The cranial parasympathetic supplies the heart and the gut with its outgrowths thus is motor and secretory to the alimentary canal and constrictor to the pupil (third cranial nerve via ciliary ganglion and short ciliary nerves). The sacral parasympathetic is a mechanism for emptying i.e. motor for bladder rectum and erection of penis. The sacral sympathetic on the other hand causes contraction of the smooth muscle in the bladder neck prostate and seminal vesicles inhibition of peristalsis in the lower colon contraction of internal anal sphincter and vasoconstriction.

All the vasomotor fibres of the body leave the cord between T 1 and L 3 and all inhibitory nerves of gut arise from the same region thus paralysis of this part of the cord will result in total vasomotor paralysis with low blood pressure and contraction of the alimentary canal (Paralytic ileus may be due to excessive sympathetic stimulation and may be cured by blocking of the anterior roots).

**FUNCTIONS OF SYMPATHETIC SYSTEM —**

- 1 Inhibitory fibres to smooth muscle of alimentary canal and constrictor fibres to the sphincters
- 2 Vasoconstrictor fibres to vessels
- 3 Some vasodilator fibres to vessels
- 4 Accelerator and augmentor fibres to the heart
- 5 Secretory fibres to sweat glands
- 6 Pilomotor fibres
- 7 Dilator fibres to bronchial tree
- 8 Dilator fibres to coronary vessels
- 9 Constrictor fibres to the spleen
- 10 Dilator fibres to the pupil
- 11 Secretory fibres to the suprarenal gland (medulla)
- 12 Inhibitory fibres to bladder wall and constrictor fibres to internal urinary sphincter fibres to other pelvic viscera

Paravertebral block is an easy way of blocking the vasoconstrictors to the limbs especially as in the upper limb. Horner's syndrome (first thoracic stellate ganglion) gives objective evidence of success. In addition vasoconstrictor impulses can be removed by (a) ganglionic blocking agents (b) reflex heating (if arm is heated leg vessels dilate and vice versa) (c) general anaesthesia especially cyclopropane.

**Effects of Drugs on Autonomic System —**

- 1 Sympathomimetic drugs e.g. adrenaline ephedrine methylamphetamine noradrenaline methoxamine and phenylephrine
- 2 Adrenergic blocking agents the antagonists of adrenaline e.g. ergot alkaloids benzodioxanes (piperovan) dibenamine di-benzylamine tolazoline (prisol) phentolamine

- 3 Parasympathomimetic drugs e.g. acetylcholine the anticholinesterases e.g. neostigmine edrophonium physostigmine  
 Acetylcholine has a muscarinic action corresponding to parasympathetic stimulation (together with vasodilatation and stimulation of sweat glands) After muscarinic action has been paralysed by atropine larger doses of acetylcholine cause a nicotinic effect—stimulation of autonomic ganglia stimulation of voluntary muscle fibres stimulation of adrenaline secretion by suprarenal medulla the classical nicotinic effect of pharmacology The nicotinic action can be abolished by larger doses of nicotine and by quaternary ammonium compounds In addition mechoyl has a muscarinic action and carbachol both a muscarinic and a nicotinic action
- 4 The cholinergic blocking drugs—the antagonists of acetylcholine acting on —
  - a The autonomic ganglia drugs antagonizing its nicotinic action e.g. nicotine in excess tetraethyl ammonium halides hexamethonium
  - b The striated myoneural junction e.g. d-tubocurarine decamethonium
  - c The plain muscle glands and heart drugs antagonizing the muscarinic effects of acetylcholine e.g. atropine scopolamine and their synthetic congeners

### NOTES ON ADRENERGIC DRUGS

#### Adrenaline (Epinephrine) —

**HISTORY** —Isolated by Oliver and Schafer in 1895 and produced in crystalline form by Takamine and Aldrich independently in 1901 the first hormone to be synthesized

**PHARMACOLOGY** —General effects similar to those resulting from stimulation of adrenergic nerve-endings but ultimate action is chiefly on effector cells It is formed in the suprarenal medulla and at adrenergic nerve endings and is excreted after degradation in the urine and destroyed by the enzyme amine oxidase and the catechol oxidases Ampoules of adrenaline can be autoclaved once or twice without loss of potency \*

**CARDIOVASCULAR SYSTEM** —Its main effect is to increase the stroke volume rate and cardiac output It increases the incidence of arrhythmias by making the automatic conducting system more irritable Adrenergic blocking agents do not prevent direct stimulation of myocardium although they do prevent arrhythmias The systolic blood pressure rises but the diastolic is not greatly altered After the blood pressure rises there comes a short period of hypotension the drug has a biphasic reaction causing secondary vasodilatation e.g. in the nasal mucosa following constriction due to adrenaline Vessels in different situations react to adrenaline in different ways While the vessels of the skin mucosæ subcutaneous tissues muscles and kidneys are constricted those in muscles and splanchnic area are relaxed While the cerebral and



**Adrenergic Drugs continued**

pulmonary arteries are constricted the coronaries vary angina pectoris may be precipitated in patients with coronary disease because of the augmentation of cardiac work on top of narrowed vessels The overall action of adrenaline on vessels is one of dilatation the increase in cardiac output being responsible for the rise in blood pressure The peripheral venous pressure is increased Following intravenous infusion the effects of adrenaline last longer than do those of nor adrenaline

**THE RENAL BLOOD FLOW**—This is decreased because of the constriction of the renal vessels

**THE RESPIRATORY SYSTEM**—The bronchi are relaxed following both topical (1 to 100 solution in atomizer) and systemic administration This may increase the tidal volume even in normal patients Depth of respiration slightly increased and irregular breathing sometimes seen If a large dose is injected the rise in blood pressure may produce a reflex from the carotid and aortic pressoreceptors causing temporary apnoea

**THE ALIMENTARY CANAL**—While the muscle of the gut is relaxed the pyloric and ileocolic sphincters are contracted The spleen contracts and empties its cells into the circulation The secretion of the intestinal glands is inhibited

Sweating and pilomotor activity not much stimulated in man Glycogen is mobilized from the liver giving rise to an increase in the blood sugar level There is very little stimulating effect on the central nervous system Large doses stimulate small clinical doses inhibit uterine tone It elevates the pain threshold Adrenaline is a mild ganglionic blocking agent but does not cause increased secretion of the adrenal cortex even though it may result in a short period of eosinopenia after injection

Following injection untoward reactions may include anxiety restlessness throbbing headache vertigo pallor and palpitations hyperthyroid and hypertensive patients are specially liable to these Nitrites should be used to combat grave hypertension

Discoloration of adrenaline indicates decomposition

Its use is contra indicated when the patient is inhaling chloroform cyclopropane or trichlorethylene because of the risk of the production of ventricular fibrillation In the presence of thyrotoxicosis and of hypertension it must be given with great caution

It is mostly degraded by conjugation with glycuronic and sulphuric acids and excreted in the urine A smaller part is oxidized by amine oxidase

**1 Noradrenaline (1 Arterenol, Levophed)** (nor=nitrogen ohne radikal—German)—A primary catechol amine differing from adrenaline in having no methyl group attached to its nitrogen atom Concerned in the humoral transmission of adrenergic nerves Is unlikely to be a precursor of adrenaline to be

synonymous with Cannon's sympathin E. It is formed at adrenergic nerve endings where most of it is destroyed by amine oxidase and the catechol oxidases very quickly so it is not suitable for adding to local analgesic agents or to saline to produce local ischaemia. Fresh human suprarenal glands contain four times as much adrenaline as noradrenaline.

**HISTORY**—Humoral transmission of nerve impulses described by Elliott in 1905. Properties of racemic noradrenaline described by Barger and Dale in 1911. Modern interest in the drug stimulated by von Euler (1946) who showed that noradrenaline is present in extracts of sympathetic nerves and Peart (1949) who showed that noradrenaline is liberated when a sympathetic nerve is stimulated. The *lævo* isomer is the more active.

**PHARMACOLOGY**—Probably the major pressor amine found at post ganglionic adrenergic nerve endings responsible for reflex vascular effects adrenaline being mainly responsible for metabolic activities. It is excreted like adrenaline.

**CARDIOVASCULAR SYSTEM**—No influence on cardiac output. In animals and man\* dilates coronary vessels. The most powerful vasoconstrictor in common use acting on all vessels except possibly the coronaries. The blood pressure both systolic and diastolic rises. Heart rate sometimes slowed a reflex activated by hypertension on the pressor receptors in the aortic and carotid sinuses. It may precipitate arrhythmias. After hypotension produced by hexamethonium salts the heart rate is quickened.

**RENAL BLOOD FLOW**—Reduced owing to constriction of renal vessels. It stimulates uterine contractions unlike adrenaline which in clinical doses inhibits them.

The *dextro* isomer acts primarily on the smooth muscle of the bronchi the *lævo* isomer acts primarily on the smooth muscle of the vessels. The drug is less toxic than adrenaline. While adrenaline is the flight or fright hormone noradrenaline is the pressor hormone. Its pressor activity is potentiated by the alkaloids of ergot: ergotamine, ergotovine and ergometrine (ergonovine).

It is put up as the bitartrate in 1-1000 solution each ml containing 1 mg of noradrenaline. It is to be given diluted by intravenous infusion. For direct injection a special solution 1:10,000 can be used in a dosage of 50-75 microgrammes ( $\frac{1}{2}$ - $\frac{3}{4}$  ml).

**INDICATIONS**—If 2 ml of 1-1000 solution are added to 500 ml of normal saline dextrose or blood the resulting solution contains 4 µg per ml. This solution has been used as a continuous intravenous drip starting at a rate of 2 ml per minute (8 µg) (microgrammes). The speed is later adjusted according to the blood pressure reading. The solution should be made up freshly for each case and obviously the reduction in the rate of drip must be gradual and must be controlled by frequent blood pressure readings. Extravasation of the drip fluid into the tissues has caused necrosis.

\* Euler U S von, *Lancet* 1955 2 151

SUMMARY OF PHARMACOLOGY OF ADRENALINE NORADRENALINE AND  
METHYL AMPHETAMINE

Drug	CARDIAC OUTPUT	SYSTOLIC BLOOD PRESSURE	DIASTOLIC BLOOD PRESSURE	PULSE RATE	MUSCLE BLOOD FLOW	SKIN BLOOD FLOW	SPLANCHNIC BLOOD FLOW	PERIPHERAL RESISTANCE	CENTRAL NERVOUS EFFECTS	RENAL BLOOD FLOW
Adrenaline	Very much increased	Increased	Unchanged sometimes reduced	Increased	Increased	Reduced	Prob bly increased	Reduced	Stimulated	Reduced
Nor adrenaline (Levophed)	Unchanged or reduced slightly	Increased	Increased	Slowed regular	Very much reduced	Reduced	Reduced	Very much increased	None	Reduced
Methyl amphetamine (Methedrine)	Increased	Increased	Unchanged or moder- ately in- creased	Unchanged or slowed	Increased	Reduced	Probably increased	Reduced	Very much stimulated	Increased

Should be run directly or via a plastic catheter into a large proximal vein to prevent tissue sloughing. Blanched and ischaemic skin areas can sometimes be prevented from sloughing by injection of acetylcholine 100-200 mg using a fine needle or of phentolamine solution 5 mg in 10 ml of saline. Once a drip is started it must not be stopped suddenly as the drug depresses the transmission through the sympathetic ganglia of impulses which maintain vascular tone and before the infusion is finally stopped transmission must be given time to recover.\*

- 1 Following removal of chromaffin cell tumours such as pheochromocytomata.
- 2 Following thoracolumbar sympathectomy especially on the second side
- 3 During operations on patients with coronary or cerebral ischaemia to maintain the blood pressure at near normal levels. In the former arrhythmias may occur and may require E.C.G. monitoring. Unlike adrenaline it causes no cerebral stimulation. It causes some cerebral vasoconstriction.
- 4 To counteract blood pressure fall after the too rapid injection of intravenous barbiturates 0.25-1 ml of the 1:10 000 solution should be used.
- 5 To sustain the circulation after massive haemorrhage while waiting for blood and possibly to enable economy in the amount of blood transfused in severe shock.
- 6 To maintain the circulation during spinal intradural and extradural analgesia. For although the chief fall in pressure in these cases is due to reduced cardiac output there is at the same time reduced peripheral resistance.
- 7 To reverse grave hypotension caused by ganglionic blocking drugs.
- 8 To counteract the hypotension associated with the continuous intravenous procaine drip especially in heart surgery. As it raises the aortic pressure and probably dilates the coronary vessels it is a potent cardiac stimulant.
- 9 In cases of peripheral circulatory collapse. Its place here is not yet determined. Infusion of toxic doses may cause acute pulmonary oedema. The vessels gradually lose their responsiveness to physiologically released or to artificially infused noradrenaline during periods of prolonged low blood pressure so active resuscitation must be started early with this drug if it is to be used at all. Should not be used in patients with oliguria as it reduces renal flow.
- 10 To treat the shock associated with coronary thrombosis.
- 11 To raise blood pressure and stimulate the myocardium thereby in cases of cardiac standstill †. The special solution 1:10 000 should be used (0.25-0.75 ml).
- 12 It is superior to methyl amphetamine in restoring the blood pressure after the administration of phenothiazine derivatives.

Burn, J. H. *Br J Anaesth* 1957 28 459

† McMullan, I. K., R. Cockett, F. B. and Styles, P. *Tkavar* 1952 7 205

## Adrenergic Drugs continued

**CONTRA INDICATIONS**—Hypertensive patients are especially sensitive to the effects of the drug and so the speed of drip must be slow until the patient settles down. Cyclopropane must be given very cautiously and never to deep planes of anaesthesia while the drug is being infused.

The reader is referred to articles by H C Churchill Davidson (*Brit med J* 1951 2 155) H J C Swan (*Ibid* 1952 1 May) H C Churchill Davidson and H J C Swan (*Anaesthesia* 1952 7 4) U S von Euler (*Lancet* 1955 2 151)

**CHEMICAL TRANSMISSION OF NERVE IMPULSES —**

In the autonomic nervous system excitation is transmitted from pre to post ganglionic nerves by acetylcholine while the post ganglionic terminations liberate acetylcholine (cholinergic fibres) or adrenaline (adrenergic fibres). All parasympathetic post ganglionic fibres are cholinergic and most sympathetic post ganglionic fibres are adrenergic (exceptions fibres to sweat glands and to vasodilators).

- 1 **ADRENERGIC**—Adrenaline and noradrenaline are liberated at nerve endings of adrenergic nerves (Barger and Dale 1910). Destroyed by amine-oxidase which is itself inhibited by ephedrine. Post ganglionic sympathetic to heart gut and vasoconstrictors i.e. all these fibres except those to uterus sweat glands and vasodilators. The adrenal medulla and the terminations of the post ganglionic fibres of the thoracic lumbar (sympathetic) division of the autonomic system produce neurohormones simulating adrenaline and nor adrenaline.
- 2 **CHOLINERGIC** (acetylcholine)—These include all nerve fibres which release acetylcholine at their terminals.
  - a Pre ganglionic parasympathetic fibres which end like pre ganglionic sympathetic fibres (d) in autonomic ganglia
  - b Post ganglionic parasympathetic fibres
  - c Post ganglionic sympathetic fibres to uterus and sweat glands (anatomically sympathetic but functionally cholinergic) and vasodilators (antidromic fibres in posterior nerve roots supplying skeletal muscle)
  - d Pre ganglionic sympathetic fibres including the splanchnic fibres to the medulla of the suprarenals
  - e Motor fibres are also cholinergic impulses being inhibited by curare

**REGULATION OF HEART'S ACTION —**

**THE CARDIAC OUTPUT**—Determined by —

- 1 **The Venous Return** which depends on the following (a) The venous tone controlled by vasomotor nerves and chemical factors such as blood oxygen and carbon dioxide tension and amount of adrenaline and noradrenaline in the blood (b) Negative pressure in the thorax during inspiration (c) Contraction of the diaphragm causing descent and squeezing out of blood from the abdomen towards the

heart (d) Tone of the arterioles capillaries etc which may contain much or little blood (e) Gravity

- 2 *The Force of the Heart beat* which depends on (a) The length of the myocardial fibres at the beginning of each systole (b) The duration of diastole more blood being pumped out the longer the diastole (c) The coronary blood flow depending on the aortic diastolic pressure and coronary tone (vagus is vasoconstrictor sympathetic is vasodilator) chemical influences are also important e.g. blood oxygen and carbon dioxide tensions

### 3 *The Cardiac Rate*

- 4 *The Arterial Blood pressure* which influences the output of the heart through effect on venous return and venous pressure

**RATE**—Bears a direct relationship to the basal metabolic rate

*Increased by*—

- Muscular exercise because of venous (auricular) reflex of Bainbridge rise of carbon dioxide tension etc
- Emotional excitement
- Environmental temperature
- Hæmorrhage and surgical shock
- Fever cardiac arrhythmias and hypoxia
- Also increased during digestion

*Decreased during sleep*

Influenced by action of (1) Nerves (2) Chemical agents

#### 1 *Nerves*—

- a *The vagus* is inhibitory (Weber 1845) Pre ganglionic efferent fibres originate in dorsal nucleus of vagus and end in the intrinsic cardiac ganglia. Post ganglionic fibres travel along the coronary vessels. Its cardiac branches are given off in the neck just below the origin of the superior laryngeal nerve but a few leave nerve in thorax so vagal stimulation there can cause vagal cardiac effects. Vagal effect resembles that of excess potassium in blood. It is abolished if high doses of atropine are given e.g.  $\frac{1}{2}$  gr after which rate rises to 150 per minute. Fibres from right vagus terminate near sino auricular node those from left vagus near auriculoventricular node. Vagi exert their effect on auricular muscle and not directly on the ventricle. Slowing or stopping of ventricles is in direct effect due to auricular slowing or to block of conduction along A V bundle.

Increased vagal activity can lead to generalized cardiac depression with decrease in rate of pacemaker force and duration of systole reduced and refractory period lessened. On conductive system vagal stimulation causes successively (1) Increase in P R interval (2) Partial A V block (3) Complete A V block. Bundle branch block has also been demonstrated.

By decreasing cardiac output hypotension may be caused but this in turn through the aortic and

Adrenergic Drugs *continued*

carotid sinus mechanisms will result in sympathetic stimulation and tachycardia—the so-called vagal escape phenomenon

On the heart vagal effect is more pronounced than sympathetic effect

- b The *sympathetic* is accelerator and augmentor an effect described by von Bezold in 1863. Fibres arise in the lateral horn cells of the cord in segments T 1–5 and as white rami enter the lateral sympathetic chain from which post ganglionic fibres pass to the heart directly via the thoracic cardiac nerves. The superior cervical sympathetic cardiac nerves pass behind the carotid sheath and on each side are joined by the superior cervical cardiac branches of the vagus. The thoracic cardiac sympathetic nerves come from T 2 to T 5. In addition to accelerator efferent fibres they contain afferent pain fibres. Other cardiac pain fibres run in the inferior and middle cervical cardiac sympathetic nerves. Others ascend to the upper middle and lower cervical ganglia from which post ganglionic fibres pass to form the upper middle and lower (cervical) cardiac nerves. On the right side these terminate in the sino auricular node on the left side in the auriculoventricular node and bundle. Stimulation of sympathetic causes increase in the rate and force of both auricles and ventricles unlike vagal stimulation which affects only the auricles and junctional tissues. Arrhythmias even ventricular fibrillation may follow sympathetic stimulation especially during chloroform cyclopropane or trilethyl anæsthesia.

Sympathetic stimulation causes (1) Dilatation of coronary vessels (2) Increase of heart rate (3) Increase of force of contractions (4) Facilitation of conduction (5) Increased irritability

Sensation is through afferents having their cell stations in the posterior root ganglia of the upper thoracic nerves—especially the second—and running in the cervical (especially the middle and inferior) and thoracic cardiac nerves thence entering cord via white rami of the upper six thoracic nerves

Reflex slowing can be caused by pressure on the lateral part of the eyeball (oculocardiac reflex) or by stimulation of nasal branches of the fifth nerve

Stimulation of afferent fibres of the vagus in the lungs as by a too strong chloroform vapour can cause reflex inhibition of heart while stimulation of the structures in the abdomen can cause extrasystoles or bradycardia independently of anæsthesia

- c The *sinus and aortic nerves*. Increase of pressure in the carotid and aortic sinuses produces bradycardia. Decrease of pressure here produces tachycardia.

**Marey's Law** Heart rate is inversely proportional to blood pressure

**The Bainbridge Reflex** (the auricular or venous reflex)  
Heart rate varies directly with venous return to auricle. Pressoreceptors are present in the walls of the right auricle and the venæ cavae. Afferent pathways of this reflex are probably in the vagus. The regulation of cardiac rate and output occurs mainly through reduction of vagal tone.

- 2 **Chemical Agents**—May be (a) drugs (b) inorganic constituents (c) acid metabolites
  - a Atropine antagonizes acetylcholine liberated at post ganglionic terminals and so depresses vagal action. Muscarine has opposite effect imitating that of vagal stimulation. Pilocarpine physostigmine acetylcholine are similar in effect to muscarine and their action is antagonized by atropine. Nicotine first stimulates then depresses the vagus it affects the ganglion cell.
  - b Calcium increases contractility and prolongs systole. Potassium reduces contractility and prolongs diastole. Sodium is necessary for both systole and diastole.
  - c Carbon dioxide and lactic acid have effect on cardiac and vasomotor centres and heart muscle. Mild excess causes tachycardia gross excess bradycardia.

**Tachycardia.**—This may be (1) A speeding up of the normal sinus rhythm (2) Supraventricular paroxysmal tachycardia (3) Ventricular paroxysmal tachycardia (4) Auricular fibrillation (5) Auricular flutter

**THE CORONARY CIRCULATION**—(1) **Nerves** The sympathetic causes dilatation the vagus constriction when stimulated. The coronary vessels fill during cardiac diastole during systole the blood is squeezed out of them. (2) **Blood Gases** A reduced oxygen tension increases blood flow a raised carbon dioxide tension increases it. (3) **The Aortic Blood pressure** A fall in this reduces coronary flow. (4) **The Cardiac Output** If this increases the coronary flow also increases. (5) **In Exercise** Violent exercise causes a great increase in blood flow. (6) **Drugs** (a) Constrictors are pituitrin (b) Dilators are adrenaline xanthine derivatives papaverine atebaine ephedrine amyl nitrite sodium nitrite nitroglycerin.

Acute hypoxia cerebral anaemia and increase of intracranial pressure all stimulate the vasomotor centre directly and raise the blood pressure.

**VARIETIES OF NERVE FIBRES**—Somatic afferent pathways begin from nerve endings in the skin etc. which are sensitive to pain touch temperature etc. Impulses are conveyed along fibres of three main types which vary in cross section and conduction rate. The smaller the fibre the slower the conduction rate. Cold also slows the rate of impulse conduction. Three main types (Gasser and Erlanger) —



**Adrenergic Drugs—Tachycardia continued**

- A Fibres** All medullated somatic nerve fibres of various diameter (1–20  $\mu$ ) Rate of conduction rapid Skeletal motor fibres touch proprioceptor and some pain and thermal fibres
- B Fibres** Medullated autonomic fibres i.e. pre ganglionic fibres e.g. white ramus Diameter 1–3  $\mu$ .
- C Fibres** Non medullated fibres both somatic and autonomic All post ganglionic sympathetic motor fibres (grey ramus) and some pre ganglionic Some afferents conveying pain and heat sensation Diameter less than 1  $\mu$  Visceral afferents are C fibres Compression blocks A fibres before C fibres while local analgesics block C fibres before A fibres
- Anoxia lowers threshold of pain fibres Stimulation of C fibres may cause a fall in blood pressure
- Visceral afferent fibres converge to the posterior root ganglia where their cell stations lie and from these central prolongations run up to the posterior horn grey matter some of them first traversing Lissauer's tract In the grey matter of the posterior horn synapses occur and the second neurone fibres cross over in the ventral commissure and ascend as the lateral spinothalamic tract to the thalamus Here after a second synapse many of the fibres go to the post central gyrus of the cerebral cortex

**CHAPTER III****PRE-ANÆSTHETIC CARE AND PREPARATION**

The patient should be seen before the day of operation Written consent for operation and anæsthesia should be obtained and the patient's wishes as to anæsthetic agent and technique should be consulted It is rarely wise to force an unwelcome technique on a patient A physical examination must be made unless it has been recently carried out by someone else of probity and experience A suitable hypnotic should be given the night before operation such as pentobarbitone gr 1½–3

Before the anæsthetist sets to work he must satisfy himself that he is dealing with the right patient he must also see that dentures are removed and the stomach empty and that permission for operation has been given in writing This is especially important in emergency operations and in operations performed on patients under 21 years of age

The following points must be investigated —

- 1 HISTORY OF PREVIOUS ANÆSTHESIA
- 2 TEMPERAMENT OF PATIENT —The anxious type will require more careful handling than the phlegmatic or placid type
- 3 HABITS OF PATIENT —Concerning alcohol—alcoholic patients are often resistant to anæsthetics concerning smoking and the bronchial catarrh it causes concerning mode of life occupation etc

4 ANATOMY —Of jaws nose teeth superficial veins etc Choice of airway depends partly on this Intermittent thiopentone may not be successful if the veins are very small Examination may show spinal analgesia or extradural lumbar or sacral block etc to be inadvisable

5 HISTORY OF PREVIOUS ILLNESSES

6 GENERAL PHYSICAL STATE

7 PHYSICAL EXAMINATION —

a RESPIRATORY SYSTEM —Presence of productive cough is more important than vague physical signs Postural drainage may be desirable before anaesthesia Perhaps accompanied by bronchodilator sprays The nose and throat should be cleared before induction An X ray of the chest may be useful in abdominal and thoracic cases

b CARDIOVASCULAR SYSTEM —The degree of exercise tolerance gives most useful information History of angina of effort or presence of decompensation cardiac enlargement arrhythmia and diastolic murmurs are important Blood pressure should be taken The percentage of haemoglobin should be assessed and cross grouping of blood arranged for should it be necessary In serious cases a slow drip should be set up so that fluid can be given easily at any time

c URINARY SYSTEM —The bladder should be emptied before patient leaves his bed Tests for sugar albumin and ketone bodies should be made In elderly males the prostate and renal functions should be investigated and a blood urea estimation performed

d BUILD OF PATIENT —If the abdomen is dome shaped the bowels will tend to pout when the peritoneum is opened Such patients if strong and fit may do well with extradural or spinal analgesia for abdominal operations Obesity may make the anaesthetic more difficult

e NUTRITION —If the state of nutrition is bad plasma protein and urinary chloride estimations are useful If value of former is less than 5 g per 100 c c latent oedema may occur Severe injuries e.g. surgical operations are accompanied by much tissue breakdown possibly so that the amino acid methionine can be liberated to aid repair This can be made less serious by storing up protein beforehand by increased amounts of eggs skimmed milk etc Thus healing can be aided wound oedema prevented and the weight loss following most operations can be mitigated Hypoproteinaemia increases the toxicity of thiopentone chloroform and cyclopropane but not of ether  
✓ High carbohydrate feeding is always wise before operation to increase glycogen reserves

Factors predisposing to avitaminosis include (i) Inadequate intake (ii) Poor absorption (iii) Destruction in gut (iv) Decreased utilization (v) Increased requirements  
Vitamin A —This is absorbed from the gut in combination with bile acids and may need supplementing in cases of obstructive jaundice in doses of 25 000 to 50 000 units daily

Pre anæsthetic Care and Preparation *continued*

**Vitamin B**—Aneurine (thiamine) aids appetite and gastric motility therapeutic dose 10 to 50 mg

Nicotinic acid (niacin) or its amide should be added if sulphaguanidine or its substitutes are used dose 200 to 500 mg

Riboflavin is useful in cheilosis dose 5 to 50 mg

**Vitamin C**—Ascorbic acid required for formation of collagen in wound healing dose 200 to 500 mg per day

✓ **Vitamin A** (analogues menaphthone BP menadione USP)—Needed by liver in formation of prothrombin. Should be supplemented in cases of obstructive jaundice pyloric obstruction and chronic diarrhoea dose 10 mg of the synthetic analogue hypodermically for several days before operation

✓ **f DEHYDRATION**—This may occur in cases of persistent vomiting e.g. pyloric obstruction or high intestinal obstruction in persistent diarrhoea etc. It should be treated by intravenous infusion of one part normal saline and four parts glucose (5 per cent)

In non urgent cases a suitable aperient should ensure a bowel evacuation on the day preceding operation. If this has not occurred a soap and water enema can be given on the evening before operation. The dehydration consequent on excessive purgation and starvation must be avoided but diet on the day before operation must be light unlikely to cause intestinal gas and easily digested. Apart from sweets, food should not be taken during the six hours preceding operation

**Posture during Operation —**

**TRENDELENBURG POSITION** \*—It is impossible even by over ventilation to prevent accumulation of carbon dioxide in the steep Trendelenburg position †. In short stout patients especially if there is an abdominal mass pressure on the diaphragm from the bowel may produce cyanosis and dyspnoea and reduction of vital capacity by 15 per cent unless intermittent positive pressure respiration is carried out. Cyanosis also occurs in the face and neck of plethoric patients in this position as a result of stagnant hypoxia due to gravity even in the presence of adequate ventilation. If the arm is abducted, the elbow should be slightly flexed and pronated to prevent pull on the brachial plexus, but every care should be taken to avoid this position

Position can be maintained by (1) Shoulder supports (with risk of brachial plexus lesions) (2) Tying ankles and thighs to operating table (with risk of phlebothrombosis of veins of calf) (3) Apparatus to support the iliac crests (Ogier Ward Hans) (4) Langton Hewer's corrugated mattress ‡

Friedrich Trendelenburg (1844–94) first used this tilt in 1890 when professor of surgery at Rostock. This was popularized by his pupil Willy Mey in 1884 in the US. Trendelenburg later occupied the surgical chairs at Bonn and Leipzig. He described a tracheotomy tube with inflatable cuff in 1869.

† Lucas B G B and Miles E H *Poc R Soc Med* 1953 46 358

‡ Hewer C L *Lancet* 1953 1 5

Great care is needed to prevent pressure on nerves or stretching of nerve trunks

✓Circulatory depression and drainage of sputum from the lungs are both aided in this position

✓ PRONE POSITION —A pillow should be placed under each shoulder and another under the pelvis, so that breathing is not unduly interfered with and so that all pressure is completely removed from the abdomen and its large venous channels. Obese patients stand this position badly. Anæsthetized patients stand rough movement badly

✓LITHOTOMY POSITION —If the patient is arranged while supine so that the anterior superior iliac spines are on a level with the break in the table he will be in good lithotomy position when the legs are supported on the stirrups and will need no further pulling about with its risk of dislodgement of intravenous needles

✓LATERAL POSITION —This handicaps the patient's breathing and a bridge makes it still worse, so it should be used as little as possible and for a short time only.

✓SUPINE POSITION —Arms should be secured by (1) A roller bandage immediately under buttocks over forearms and tucked again under buttocks (2) Wrist straps which are attached to a strap encircling table or to the table directly

Arms should not be secured by placing hands under buttocks because of risk of pressure on them. Nor should arms be kept raised above head. If intravenous therapy is required the arm should be supported on an arm board well padded to prevent arm from being displaced behind the coronal plane of the body. Better still the arm should be at the patient's side and an extra length of rubber tubing incorporated into the drip apparatus. Pressure should be taken off the veins of the calf by supporting the heels on soft pads

Tidal exchange is handicapped in various positions assumed during operation. It has been estimated that compared with the sitting position tidal exchange is decreased 9 per cent in reversed Trendelenburg position 10 per cent in supine prone and left lateral positions 12 per cent in right lateral position 13 per cent with use of gall bladder bridge 15 per cent with Trendelenburg position 18 per cent with lithotomy position. Clinically the gall bladder and the kidney bridges definitely handicap the patient and reduce the blood pressure

## CHAPTER IV

THE PHARMACOLOGY OF DRUGS USED FOR  
PRE-OPERATIVE AND POST-OPERATIVE  
MEDICATION

The administration of drugs to ease the induction and maintenance of anaesthesia by —

- ✓ 1 Reducing the metabolic rate and minimizing fear and anxiety
- 2 Minimizing the secretions of the salivary glands and upper respiratory tract

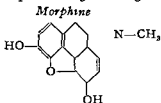
May be —

- ✓ a Sedative (barbiturate bromethol morphine methylpentynol ataraxic drugs phenothiazine derivatives)
- ✓ b Analgesic (morphine pethidine opium)
- ✓ c Parasympatholytic (atropine scopolamine)

Sedative action is helped if a suitable hypnotic is given the night before operation

Examples of hypnotics are —

- a Soluble barbitone (medinal) 5–10 gr
  - b Pentobarbitone (nembutal)  $1\frac{1}{2}$ –3 gr (100–200 mg)
  - c Quinalbarbitone (econal)  $1\frac{1}{2}$ –3 gr
  - d Phenobarbitone (luminal) 1 gr
- ✓ If patient is in pain pethidine 50–100 mg may be added



- ✓ **Morphine** — (From the Greek Morpheus god of dreams) Has been in use as opium for over 2000 years and is still the best available analgesic (Among the remedies which it has pleased Almighty God to give to man to relieve his sufferings none is so universal and so efficacious as opium — Thomas Sydenham 1680 and still true in 1958) Obtained from the seed capsules of the poppy plant

Morphine isolated from opium by Serturmer in 1806 Morphine salts are not destroyed by boiling

First used as pre anaesthetic drug by Lorenzo Bruno of Turin in 1850 and by Claude Bernard in 1869

One of over 25 alkaloids contained in opium (*Papaver somniferum*) but only morphine codeine and papaverine have wide clinical use Concentration of morphine in opium is 10 per cent

**PHARMACOLOGY** — Morphine is a direct metabolic depressant. Its chief effects are on the central nervous system, the respiratory system and the bowel. Has been synthesized by Gates and Tscudi (1952). Like codeine, heroin and diacetylmorphine it is related to phenanthrene and piperidine, this group of drugs being good analgesics and poor relaxers of smooth muscle. Papaverine is related to benzylisoquinoline and like its congeners is a poor analgesic but a good relaxant of smooth muscle. Morphine may release histamine from the tissues.

**CENTRAL NERVOUS SYSTEM** — It produces analgesia and sleep, the former predominating. In addition there is euphoria and emotional placidity and muscular relaxation which may outlast the analgesia. Very rarely stimulation results causing restlessness and delirium, this is the normal reaction of the cat and horse to morphine. Spinal cord reflexes may — rarely — be exaggerated and children have been seen in spinal convulsions after morphine. Single doses have no marked effect on the E.E.G.

It differs from the barbiturates as follows —

- a Its analgesic effect is greatly superior
- b It is a poor anticonvulsant
- c Its depressing effects on breathing are greater

It contracts the pupil by a central, not a peripheral action, by stimulating the pupillary constrictor fibres in the Edinger Westphal nucleus. Tonic impulses to the ciliary muscle travelling along the oculomotor nerve. Intra-ocular and cerebrospinal fluid pressure is raised.

Analgesia is more efficient if given before the onset of pain than if given to relieve existing pain. With sufficient dosage analgesia sufficient to perform surgical operations can be produced but only at the expense of severe depression of respiration. Has no local pain relieving effect.

Intravenous injection causes a more rapid, less prolonged and less intense effect than intramuscular injection. Analgesic effect is directly proportional to dose.

**RESPIRATORY SYSTEM** — There is a direct depressing effect on the respiratory centre causing a diminution in both rate and depth of breathing and a smaller minute volume. This is seen even in very small subhypnotic doses. With large doses respiratory failure and ultimate arrest occur, this is how morphine kills and is due to medullary depression. Bronchial constriction may occur in asthmatics who receive morphine.

Alveolar carbon dioxide tension is increased but respiratory centre does not respond to it. Maximal respiratory depression occurs 30 minutes after intramuscular injection.

Respiratory arrhythmia (Biot's breathing) and periodic breathing (Cheyne Stokes) may be caused.

When giving ether the reduced minute volume cannot be compensated for by increasing the tension of the vapour as laryngeal irritability is not greatly depressed, this leads to coughing and laryngeal spasm.

**Morphine—Pharmacology continued**

**GASTRO INTESTINAL TRACT**—Morphine constricts the sphincters of the gut promotes glycogenolysis and so raises the blood sugar. It stimulates the antidiuretic effect of the pituitary. The movements of the stomach are reduced while the pylorus is contracted. The tone of the muscles of the small and large intestines is increased but peristalsis is reduced and so constipation results from a state of spastic immobility of the bowel. The effects of morphine on the alimentary canal are local and not central. Atropine antagonizes this action neostigmine increases it.

**Nausea and vomiting** are due to stimulation of the medullary chemoreceptor trigger zone of Borison and Wang and not due to direct stimulation of the vomiting centres. This is seen most strongly with apomorphine. In patients who are readily made sick this can sometimes be prevented by giving a small dose of morphine e.g.  $\frac{1}{4}$  gr. forty five minutes before a larger dose presumably this acts by depressing the vomiting mechanism. Vomiting after morphine depends partly on the movements of the body and the position of the patient. It sensitizes the vomiting centre to vestibular movements. Dramamine and other histamine inhibitors have a protective effect and so has chlorpromazine. Ambulation after morphine will cause more nausea than quiet bed rest.

Morphine produces a contraction of the muscle at the lower end of the common bile-duct (sphincter of Oddi) and so raises the bile pressure in the bile ducts by preventing emptying. Atropine does not fully antagonize this action but nitro glycerin nalorphine adrenaline aminophylline and amyl nitrite do (as is also the case with pethidine).

The tone and peristalsis of the ureters and other smooth muscle e.g. of the hollow viscera bladder sphincter etc. is increased an action antagonized by atropine. The tone of the Fallopian tubes is increased and spasm potentiated. The tone of the detrusor muscle and the vesical sphincter is increased and may hinder micturition.

**THE CARDIOVASCULAR SYSTEM**—Not greatly altered by clinical doses of morphine. There is sometimes a slight fall in pulse rate and blood pressure especially if drug is given intravenously. Vascular collapse may follow if a morphinized patient suddenly sits up or is moved. Sympathetic stimulants and leg bandaging will abolish this effect. There is vasodilatation of skin vessels especially in the head and neck (the blush area). Skin weals may also be seen when morphine is applied to scarified skin e.g. sometimes following an injection. Sweating may be stimulated.

Patients in shock should be given their morphine intravenously so that it does not accumulate unabsorbed in the ischaemic tissues only to produce a massive effect when absorption occurs with improvement in the shocked c. \*

Morphine sometimes causes itching especially of the nose. It may cause occasionally anaphylactoid and allergic reactions ranging from slight syncope to anaphylactic shock with bronchial asthma.

**EXCRETION**—A small part detoxicated in the liver a larger proportion being excreted by the kidneys and by the gastrointestinal tract.

It appears in breast milk saliva and sweat and readily passes the placental barrier when it may have a profound effect on the foetal respiration.

The administration of opiates may be responsible for an increase in the serum glutamic-oxalacetic transaminase activity in some individuals.\*

**SUSCEPTIBILITY TO MORPHINE**—Infants under 6 months are very susceptible and so are the aged feeble and debilitated. Children tolerate it well in doses proportional to body weight. Hyperthyroid patients tolerate morphine well hypothyroid cases badly. Patients in pain especially that due to cardiac infarction may require large doses and tolerate it well. In patients with raised intracranial pressure administration of morphine may cause dangerous respiratory depression. Chronic nephritis and uræmia are not contra indications. Patients with Addison's disease are readily depressed by morphine. Morphine may prove fatal in cases of compensatory hyperpnoea due to heart failure secondary to chronic pulmonary disease—chronic cor pulmonale. It may be life saving in the useless dyspnoea of cardiac asthma or acute pulmonary oedema.

The hydrochloride sulphate and tartrate are used while the dose in adults is usually between  $\frac{1}{4}$  and  $\frac{1}{2}$  gr (20–8 mg). An adult weighing 10 stone will get maximum pain relief with minimal side-effects on a dose of 10 mg ( $\frac{1}{4}$  gr). It can be given according to the formula  $\frac{1}{4}$  gr per stone of body weight with  $\frac{1}{2}$  gr as a maximum dose. Should be given 90 minutes before anaesthesia so that peak of respiratory depression is passed before induction commences. To make the ebbing life of a patient dying of carcinoma tolerable the following cocktail may be given and repeated as required (Brompton Hospital). Morphine gr  $\frac{1}{4}$  and cocaine gr  $\frac{1}{4}$ . Honey and gin 60 mins of each (or 60 mins of syrup and 30 mins of rectified spirit). Chloroform water to  $\frac{1}{2}$  oz.

$\left[\frac{1}{4} \text{ gr} = 16 \text{ mg} \quad \frac{1}{2} \text{ gr} = 32 \text{ mg}\right]$

**Codine**—This is methyl morphine and together with morphine and papaverine forms the chief alkaloidal derivative of opium. It was isolated in 1832 by Robiquet.

It depresses respiration less than morphine causes less constipation than morphine but is more depressing to cough reflex than morphine. Its analgesic effect is one sixth that of morphine.



## Morphine—Pharmacology continued

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Guedel states that the dose of pre anaesthetic sedative should depend on the metabolic rate of the patient. Factors which raise this are —

- 1 Fear and anxiety
- 2 Pain
- 3 Fever
- 4 Hyperthyroidism

With a high metabolic rate morphine is readily excreted

#### Advantages and Disadvantages of Morphine as Premedication —

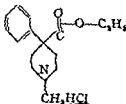
##### ADVANTAGES —

- 1 Relieves anxiety and produces tranquillity thus delirium of second stage reduced
- 2 Reduces amount of anaesthetic needed
- 3 Helps to prevent rapid and deep breathing

##### DISADVANTAGES —

- 1 May produce post-operative constipation vomiting and ileus
- 2 Causes respiratory depression and so may retard induction of inhalation anaesthesia or cause respiratory arrest when associated with cyclopropane. Nembutal does not produce these effects so strongly. Addition of carbon dioxide will speed induction but the respiratory depression may last into the post operative period and may encourage atelectasis. Morphine in addition depresses the cough reflex.
- 3 It interferes with pupil signs of depth of anaesthesia. When morphine is combined with atropine or scopolamine its miotic action usually proves stronger than their mydriatic effect.
- 4 It is habit forming.

**Pethidine** (demerol meperidine hydrochloride isonipocaine dolantin dolantal dolosal pantalgin) — The hydrochloride of the ethyl ester of 1 methyl 4 phenyl piperidine 4 carboxylic acid



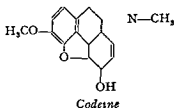
First synthesized in 1939 by Schaumann and Cisleb. Intravenous dose 5-5 mg repeated as necessary

##### PHARMACOLOGY —

- a Has a morphine like action on pain about one tenth as powerful as morphine 100 mg being the equivalent of about 10 mg ( $\frac{1}{4}$  gr) of the latter drug. Relieves most types of pain

## Codeine continued

and fails to produce progressive sedation with increasing doses  
Less likely to cause addiction than morphine Most of drug



is excreted unchanged by kidneys The usual dosage is  $\frac{1}{4}$ – $1\frac{1}{2}$  gr  
(15–90 mg) of the phosphate

**Heroin**—This is diacetyl morphine and is the most likely of the opium derivatives to become a drug of addiction because of the euphoria it creates It depresses the respiratory centre and the cough reflex more than morphine and is four to eight times as efficient as an analgesic Vomiting less common than after morphine but constipation more common

Excretion is chiefly by the kidneys

The usual dose is  $\frac{1}{4}$ – $\frac{1}{2}$  gr of the hydrochloride

**Dilaudid**—Dihydromorphinone Nausea vomiting and constipation less marked than with morphine Four times as potent as morphine as a narcotic ten times as potent as an analgesic

The usual dose is  $\frac{1}{4}$ – $\frac{1}{2}$  gr (2–5 mg)

**Papaveretum B P C** (omnupon pantopon alopon opoidine)—These are mixtures of purified opium alkaloids in the proportion found in nature They contain 50 per cent of morphine Their papaverine content is not sufficient to produce pharmacological effect There is no evidence that papaveretum causes fewer unpleasant side effects than morphine

Dosage  $\frac{1}{2}$ –1 gr (20–40 mg)

**Papaverine**—Isolated by Merck in 1848 from opium Related to isoquinoline and is different in constitution and action from morphine Possesses an anti spasmotic action in addition to its analgesic effect but does not suppress intestinal peristalsis Has an antibrillating effect on the ventricles but can cause cardiac arrhythmia and death if injected intravenously quickly Relieves spasm in arteries Has almost no effect on the central nervous system on respiration or on mood and causes no euphoria Dose up to 100 mg intravenously very slowly

**Apomorphine**—Prepared by treating morphine by strong mineral acids Not used for premedication but is derived from morphine Used to treat delirium e.g. that following cyclopropane or scopolamine in subemetic doses Apomorphine 1–2 mg in 10 ml of saline injected slowly intravenously provided that lack

is not the cause of the delirium. Can be used to empty stomach in emergency by stimulating the vomiting centre before induction of anaesthesia. Dose gr  $\frac{1}{4}$  intravenously with additional gr  $\frac{1}{8}$  if necessary. The addition of atropine gr  $\frac{1}{8}$  will prevent possible vagal effects.

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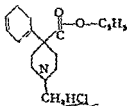
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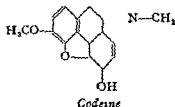
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Excretion is chiefly by the kidneys

The usual dose is  $\frac{1}{4}$ - $\frac{1}{2}$  gr of the hydrochloride

**Dilaudid**.—Dihydromorphinone Nausea vomiting and constipation less marked than with morphine Four times as potent as morphine as a narcotic ten times as potent as an analgesic

The usual dose is  $\frac{1}{8}$ - $\frac{1}{4}$  gr (2-25 mg)

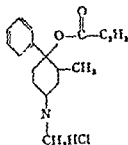
**Papaveretum BPC** (omnupon pantupon alopon opoidine)—These are mixtures of purified opium alkaloids in the proportion found in nature They contain 50 per cent of morphine Their papaverine content is not sufficient to produce pharmacological effect There is no evidence that papaveretum causes fewer unpleasant side effects than morphine

Dosage  $\frac{1}{2}$ -2 gr (20-40 mg)

**Papaverine**—Isolated by Merck in 1848 from opium Related to isoquinoline and is different in constitution and action from morphine Possesses an anti spasmodic action in addition to its analgesic effect but does not suppress intestinal peristalsis Has an antifibrillating effect on the ventricles but can cause cardiac arrhythmia and death if injected intravenously quickly Relieves spasm in arteries Has almost no effect on the central nervous system on respiration or on mood and causes no euphoria Dose up to 100 mg intravenously very slowly

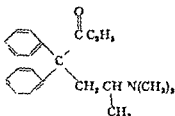
**Apomorphine**—Prepared by treating morphine by strong mineral acids Not used for premedication but is derived from morphine Used to treat delirium e.g. that following cyclopropane or scopolamine in subemetic doses Apomorphine 1-2 mg in 20 ml of saline injected slowly intravenously provided that oxygen lack

detoxicated by the liver. The effects of an intravenous dose last about 45 minutes. The recommended dose is 20-30 mg intravenously and double this by mouth. In anaesthesia it is used instead



of pethidine to supplement gas oxygen and thiopentone and appears to be a better analgesic than pethidine and to cause in proper analgesic doses less respiratory depression. It raises the pressure of cerebrospinal fluid, an effect prevented by levallorphan. Its rapid onset and rather short effect make it useful in labour for pain relief.

**Physeptone** (amidone dolophine miadone adanon butalgin methadone B.P.C.)—This is *d,l* 6-dimethylamino 4,4-diphenyl heptan-3-one hydrochloride and was studied in Germany as Hoechst 10820. A white crystalline powder soluble in water



and alcohol. An analgesic with a similar effect to that of morphine. Can be given by mouth or by injection. In common with morphine its analgesic effect is diminished if given together with hyoscine. Not a good sedative and so is not very useful as a drug for premedication unless pain is severe. Its effect in common with that of pethidine and morphine is enhanced if neostigmine 0.5 mg is given at the same time. Most useful in treatment of post-operative pain, dysmenorrhoea, pain of spasm of bladder neck, cough, renal colic, etc. Tolerance to its use is produced while it has proved to be a drug of addiction. Side effects are dryness of mouth, headache, nausea and vomiting, drowsiness and euphoria; they are less troublesome than after either morphine or pethidine. Causes bradycardia, peripheral vasodilatation and some hypotension. Has a similar effect in releasing muscle spasm as pethidine. Useful to relieve the pain associated with paralytic ileus as it does not produce the spastic inactivity of the bowel seen with morphine.

Pethidine—Pharmacology *continued*

especially those associated with plain muscle spasm—except biliary colic. Produces sleepiness but little euphoria or amnesia and so is not the ideal drug for pre-operative sedation. Somewhat uncertain in its action. Depresses respiratory centre. Is a local analgesic. Raises the CSF pressure. Can cause addiction.

- b Has a direct papaverine like effect on the smooth muscle of the bronchioles intestine ureters and arteries. In dilute solution is a vasodilator after intravenous injection. May cause hypotension. Will often relieve bronchial spasm. It causes spasm of the sphincter of Oddi: an effect counteracted by amyl nitrite nitroglycerin adrenaline and aminophylline (not by atropine or papaverine). Reduces tone and amplitude of contraction of ureters. Does not lead to constipation.
- ✓ c Has an atropine like effect on cholinergic nerve endings, causing a dry mouth dilated pupils and depression of cholinergic nerve supply to smooth muscle.
- d May release histamine from tissues producing a typical triple response causing urticarial weals over veins into which it is injected and major or minor circulatory collapse. Weal reaction is lessened if solution is 1 per cent or less in strength and is abolished if made up to 1 per cent in 0.25 per cent procaine solution. Also has antihistamine properties.
- e Has a quinidine like effect on myocardium and has been used to reduce the incidence of arrhythmia associated with cyclopropane anaesthesia.
- f Side effects may include hypotension nausea and vomiting vertigo and limb tingling. These are worse after intravenous than after intramuscular injection. Like morphine pethidine may cause hypotension if the head of the patient is raised.

**ADMINISTRATION**—Can be given intramuscularly or intravenously. Effect comes on in 15 minutes at maximum in 90 minutes and lasts up to 2 hours after intramuscular administration. May be used as an analgesic to reinforce gas-oxygen and thiopentone anaesthesia when it is given intravenously in repeated doses of 20 mg.

**EXCRETION**—Destroyed in body to extent of 80 per cent probably by hydrolysis in liver disease of which may retard its destruction. About 10 per cent is excreted unchanged by the kidneys.

**PETHIDINE IN LABOUR**—See Chapter XXVI

Congeners of pethidine include bemidone (hydroxypethidine) keto bemidone and nisental (alphaprodine).

**Nisental\*** (alphaprodine)—This is 1,3 dimethyl 4 phenyl 4 propionyloxy piperidine and was synthesized by Lee in 1947. It is structurally similar to pethidine. It does not produce tolerance on repeated injection while it causes less side effects than does pethidine. It leads to hyperglycaemia and is probably

Each teaspoonful of the elixir and each capsule contains 250 mg. The dose for an adult is 250-750 mg. for a child under 8 years 250-500 mg. It has recently been shown that it can produce toxic effects\*.

The potentiation of analgesic drugs by cholinergic agents such as neostigmine and pyridostigmine has been reported† and may be due to inhibition of destruction of the analgesic in the liver. Morphine, pethidine, levorphanol and methadone may all act for a longer time after 1 mg. of pyridostigmine or 0.5 mg. of neostigmine.

Atropine—The alkaloid of the *Atropa belladonna* or deadly night shade. Belladonna has been in use for many centuries. Atropine isolated in 1831 by Mein. Heidenhain discovered its antisalivary action in 1872. The atropine group of alkaloids are esters formed by the union of an aromatic derivative of benzyl alcohol, tropic acid with organic bases—tropine (atropine) and scopine (scopolamine). The tropic acid is the active radical. Atropine is the racemic mixture of dextro and laevo hyocyamine. Scopolamine is laevo rotatory. First synthesized by Willstätter in 1896 and again by Robert Robinson in 1917.

It has a blocking action on structures supplied by the post ganglionic cholinergic nerves i.e. smooth muscles and secretory glands acting on the effector cells. It is a parasympathetic depressant, a parasympatholytic anticholinergic drug which inhibits acetylcholine. It inhibits the muscarinic but not the nicotinic effects of acetylcholine. It raises the basal metabolic rate and in large doses potentiates the action of *d* tubocurarine chloride on the myoneural junction (Macdowall). It is active after intravenous injection. Like morphine may produce a skin wheal after subcutaneous injection.

**ACTION ON CENTRAL NERVOUS SYSTEM**—It stimulates the medulla and higher centres and directly stimulates the respiratory centre sufficiently to counteract the depressing effect of morphine. Occasionally restlessness and delirium are seen. With larger doses comes central depression. Atropine like its allies the antihistamines may cause slight drowsiness. The peak of its effect is one hour after hypodermic injection which wears off rapidly.

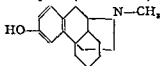
There is paralysis of the sphincter of the iris resulting in dilated pupils although a dose of gr.  $\frac{1}{16}$  does not greatly influence accommodation. The sphincter muscle is innervated from the third cranial nerve via the ciliary ganglion and short ciliary nerves. Atropine does not appreciably raise the intra ocular pressure in normal eyes but in glaucoma this definitely occurs through interference of drainage of aqueous through the channels of Fontana and the canal of Schlemm.

Sweat, bronchial and salivary glands are paralysed while bronchial muscle is relaxed causing a slight increase in the dead space. Must be used carefully in hyperthermic patients especially in children.



*Physeptone continued*

As with morphine and pethidine its side effects are worse in ambulant patients than in those confined to bed. Causes less miosis than morphine. Does not constipate. Is a mild local analgesic and antihistamine. Partly excreted in urine and partly in faeces. Powerful depressant of foetal respiration. Dosage similar to morphine—7.5–15 mg. For the relief of cough 2.5 mg in a linctus is useful.

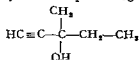
*Levorphan (Dromoran)*

**Dromoran** (laevo morphinan tartrate levorphanol levorphan methorphan) — Was synthesized by Schneider and Grisner in 1949. The laevo isomer is twice as active as the racemic drug. It is effectively absorbed when given by mouth, the dose being 1.5–2 mg which lasts 8–10 hours. It does not cause the hangover sometimes seen after morphine. In severe pain 4 mg may be needed. Does not suppress the cough reflex and may cause intestinal spasm and so is not the best analgesic for colic. Not a good pre anaesthetic drug as it neither induces sleep nor relieves anxiety. Supplied in 1 and 5 mg tablets and solution containing 2 mg per ml. Dextro methorphan (romilar) is a cough suppressant. Is 3-hydroxy-N-methyl morphinan. An analgesic effective by mouth or parenterally, more potent and of longer duration than morphine. Sleep and nausea not readily produced. Has been used as a pre or a post-operative drug. Dose 1–5 mg.

**Metapon** (methyl dihydromorphinone) — This was introduced by Eddy in 1936 as an analgesic twice as potent as morphine. Prepared from thebaine. Causes respiratory depression but rarely nausea. Can be given by mouth and so is useful for ambulatory patients e.g. those with carcinoma. An expensive drug, the dose being 6–9 mg.

**Phenadoxone** (heptalgin heptazone) — Is related to physseptone and has a weak spasmolytic effect on smooth muscle. Has fewer side-effects than morphine or physseptone but is not so potent an analgesic. Dose by injection 10 mg, by mouth 5–30 mg.

**Methylpentynol** (oblivon somnesin) — This is said to banish apprehension and anxiety without promoting sleep. It is relatively



non-toxic, has no respiratory depressant action and is rapidly metabolized. It is suitable for ambulant patients and although it induces a sense of euphoria it does not lead to incoordination.

The mortality of cases treated in this way is low. Recently coma due to excessive dosage of barbiturates has been successfully lightened by the intravenous injection of hemegride and amphenazole (see Chapter XIV).

Barbital (diethyl barbituric acid) (veronal) was first of this series to be used clinically in 1903. As pre-anesthetic sedatives barbiturates are administered by mouth or by the rectum. Three to five times the hypnotic dose usually produces coma. Suitable drugs of this type are —

1. **PENTOBARBITONE** (nembutal) — This is sodium ethyl methyl butyl barbiturate. Pentobarbitone can be administered parenterally dissolved in propylene glycol each ml containing 60 mg of the drug. The usual dose is 60–90 mg given deeply intramuscularly\*. The ordinary intravenous solution of the sodium salt in water (7.5 per cent) can also be given intramuscularly†. Pentobarbitone is rapidly and completely eliminated and does not produce cumulative effects.

It is supplied in yellow capsules containing  $\frac{1}{2}$ ,  $\frac{3}{4}$  or  $1\frac{1}{2}$  gr and also in tablets. The adult dose is 3–4½ gr given two hours before operation. The elixir contains 2 gr to the ounce. *This pentone* can be given intramuscularly in 2.5 per cent solution with hyalase. The dose is 6–8 mg per lb of body weight. Sleep comes on in 15 minutes and lasts an hour.

**QUINALBARBITONE** (sodium seconal) — Was described in 1935. This is sodium propyl methyl-carbinyl allyl barbiturate. Action is shorter, more intense and more rapid in onset than that of pentobarbital. Has been used intravenously dissolved in water and polyethylene glycol. Used similarly to intravenous pentobarbital, i.e. before intubation when it is less likely to cause laryngeal spasm than is thiopentone used during regional analgesia to produce sleep. Thioquinalbarbitone is known as thiamyl surital thioseconal.

Supplied in scarlet capsules each containing  $\frac{1}{2}$  gr,  $1\frac{1}{2}$  gr and in  $\frac{1}{4}$  gr tablets. Usual dose 3–4½ gr.

3. **BUTOBARBITONE** (sodium soneryl neonal) — Sodium butyl ethyl barbiturate. Supplied in white capsules containing 2½ gr of the drug (150 mg).

Dosage is by body weight and varies between three and five capsules. It is not excreted as quickly as nembutal.

4. **AMYLOBARBITONE** (sodium amytal) — Is sodium ethyl isoamyl barbiturate (1921). Supplied in capsules of 1 gr (65 mg) and 3 gr (200 mg). The bed time dose is 200 mg. Sodium amytal and sodium seconal in 4 per cent solution are also local analgesics.

Barbiturates antagonize the convulsant effects of local anesthetic drugs and so are suitable in the premedication of patients to be operated upon under local analgesia.

Nembutal 3 gr slightly increases the pulse rate and lowers the blood pressure. Tidal exchange and minute volume are slightly decreased. It causes less respiratory depression than morphine  $\frac{1}{2}$  gr.

Jarvis J. R. *Ohio St. med. J.* 1951 49 308  
 † Beecher H. K. *Anesthesiology* 1951 12 863

## Atropine continued

**ACTION ON CIRCULATORY SYSTEM** —Rate of heart sometimes slowed at first due to medullary (vagal) stimulation but this effect is not seen after intravenous injection of clinical doses. It may occur after hypodermic injection or after intravenous injection of small amounts such as gr  $\frac{1}{16}$ . Later rate is quickened by vagal paralysis and its effect on the S A pace maker. This increase in heart rate is not marked in infants and senile patients. Atropine has a greater effect on the right vagus which affects heart rate than on the left vagus which affects myocardial force. Atropine  $\frac{1}{4}$  gr subcutaneously will increase pulse rate 20-30 per minute effect lasting up to two hours. If  $\frac{1}{8}$  gr is given intravenously rate of heart will increase about 20 beats per minute. Reflexes involving vagal stimulation and hence cardiac slowing and syncope may be prevented by atropine.

Atropine sometimes causes dilatation of the vessels of the face. The tone and peristalsis of the gut and urinary tract are decreased. Inhibition of sweating may lead to increase in temperature. Milk secretion is not affected although the drug may be excreted into the milk. Passes placental barrier. Acts as a local anaesthetic being half as potent as procaine.

Excretion is partly by the kidneys another part being destroyed in the body. Must be used carefully in thyrotoxicosis because of action on heart rate and on B M R.

Given in adequate dosage  $\frac{1}{8}$  to  $\frac{1}{4}$  gr intravenously atropine blocks the muscarinic action of neostigmine on the heart gut and salivary glands.

Usual dose  $\frac{1}{16}$  to  $\frac{1}{4}$  gr (0.4 to 0.8 mg). gr  $\frac{1}{8}$  = 0.6 mg is rather on the small side. \*Even babies tolerate gr  $\frac{1}{16}$ .

**Scopolamine (Hyoscine)** —The laevo rotatory alkaloid is used. Isolated in 1871. Used together with morphine before anaesthesia in 1900 by Schneiderlinn. It is a better drying agent than atropine.

Actions similar to atropine. Chief difference is that scopolamine is a depressant of the central nervous system causing drowsiness, sleep and amnesia. Occasionally it produces restlessness and excitement, especially in old patients and those with unrelieved pain. With morphine it acts synergistically on cortex in opposition in medulla.

It is a mild respiratory stimulant while its action on the iris, the salivary sweat and bronchial glands is stronger than that of atropine. Has a beneficial effect on motion sickness. Tachycardia not often seen. Action on heart intestine and bronchioles is weaker than that of atropine. Is usually given with morphine in the proportion of 1 : 25.

Usual dose  $\frac{1}{16}$  to  $\frac{1}{8}$  gr (0.3 to 0.6 mg).

Combination of scopolamine  $\frac{1}{16}$  gr (0.4 mg) with pethidine 100 mg or papaveretum gr  $\frac{1}{2}$  (20 mg) forms a useful sedative before operation.

# PREMEDICATION IN CHILDREN

Children rarely need a soporific the night preceding operation. If they do nembutal  $1\frac{1}{2}$ -2 gr. is suitable.

Children under two years of age only require atropine the dose being  $\frac{1}{16}$  gr. for the smaller infants and  $\frac{1}{8}$  gr. for the larger and more robust. As children readily salivate the administration of atropine is very necessary. A dose of  $\frac{1}{16}$  gr. is suitable for a child of between 2 and 5 while over the age of 5  $\frac{1}{16}$  gr. is not too large as children tolerate atropine well.

**Morphine**—Gr.  $\frac{1}{10}$  can be given for each stone of body weight. If a  $\frac{1}{4}$ -gr. tablet is dissolved in 10 ml. of water in a syringe 1 ml. is the dose for each stone. Similarly if an ampoule containing omnopon  $\frac{1}{4}$  gr. and scopolamine  $\frac{1}{16}$  gr. is diluted to 10 ml. 1 ml. can be injected for each stone of body weight.

Morphine is badly tolerated by newly born infants but over the age of 6-12 months they do well with the drug.

For performance of Ramstedt's operation under local analgesia nepenthe 1 min. is injected hypodermically while the infant is given syrup of chloral to suck on a dummy during the operation.

**Nepenthe**—Contains 0.9% per cent of total opium alkaloids or 0.84 per cent of anhydrous morphine. For injection it is prepared in  $\frac{1}{2}$  oz. containers and the dosage is 1 min. per year in children.

**Pentobarbitone (Nembutal)**—Nembutal is well tolerated by children. By mouth the dosage can be 0.6 gr. per stone, with 3 gr. as the maximum. Seconal has a similar dosage.

By rectum, 1 gr. per year is adequate, with a maximum dose of 6 gr. The drug is given either in suppository or the capsules pierced at each end with a pin can be inserted into the rectum three hours before operation.

✓ **Chloral Hydrate**— $\text{CCl}_3\text{CH}(\text{OH})_2$ . Prepared in 1832 by Liebig and used as a soporific in 1869. It is changed in the body into trichlorethanol  $\text{CCl}_2\text{CH}_2\text{OH}$  and trichloroacetic acid. This when given by mouth is a safe and efficient pre operative sedative. Dose 5 gr. per stone body weight.

## CHAPTER V

# INHALATION ANÆSTHESIA

## THEORY OF INHALATION ANÆSTHESIA

Many theories have been put forward but so far no truly satisfactory explanation of how anæsthetics act has been produced. (See G. A. Moseley *Brit J Anæsth* 1955 27 49)

Some of the most important theories are —

1. That anæsthetics interfere with intracellular oxidation possibly by influencing enzyme action (Quastel 1941). During anæsthesia

**Paraldehyde**—A useful hypnotic in doses of one to two drachms (4-8 ml). Effect comes on thirty minutes after oral administration. Can be given in iced fruit juice or per rectum in twice the dosage with double its volume of olive oil. Its stench is its chief disadvantage. It does not depress the heart. Mostly destroyed in liver, a small amount excreted by lungs and kidneys.

**Chloral Hydrate**—A needlessly neglected hypnotic introduced in 1869 by Liebreich ( $\text{CCl}_3\text{CH}(\text{OH})_2$ ). Hypnotic dose is 15-30 gr (1-2 g) suitably diluted. Effect comes on in less than an hour and lasts about eight hours. It leaves no hang-over and is safe. A portion is converted to trichloroacetic acid and a part is excreted by kidneys after conversion to trichloroethanol by conjugation in liver with glycuronic acid to form urochloralic acid.

**Phenothiazine Derivatives**—These have been given as sedatives before operation in various doses and in (diver) combinations. They are also likely to reduce the incidence of vomiting. Chlorpromazine 25 mg the night before operation the morning of the operation and continued for several doses post operatively by mouth is a popular prescription while 300 mg as a suppository per rectum is also successful\*. Promethazine 50 mg intramuscularly with or without pethidine has been used with considerable satisfaction. To this combination a drug to produce sleep can be added e.g. an opiate or pethidine or pentobarbitone.

**Ataraxic Drugs**—(From Greek *ataraxia*=peace of mind). The tranquilizers have been used for pre operative sedation. One of these, paccatal (a phenothiazine derivative) has given good results. In addition to good pre operative sedation paccatal is said to decrease the amount of anaesthetic drugs necessary, does not alter the response to pressor drugs (unlike chlorpromazine) and reduces the incidence of vomiting. The dose suggested is 1 mg per lb body weight in adults, 1.5 mg per lb in children. Other ataraxic drugs include pipradol, atarax, ritalin, meprobamate, dioxolane, nostyn, promazine, compazine, reserpine etc.†

**PROLADONE**—This is dihydrohydroxy-codeinone pectinate and is slightly viscous in aqueous solution. It is a long acting analgesic which has been used to relieve post-operative pain and the pain due to inoperable carcinoma. It is said to cause less nausea and respiratory depression than morphine. The usual dose is 10 mg intramuscularly. The intravenous route of administration is not recommended.

**DIHYDROCODEINE BITARTRATE (D.F. 118)†**—This drug in 30 mg doses is a very powerful analgesic, remarkably free from unpleasant side effects such as nausea and respiratory depression. Duration of pain relief after hypodermic injection is about four hours. Put up in ampoules of 50 mg in 1 ml the solution has a pH of 3.2 so that it should be diluted before intravenous injection. It releases histamine. It can be given intravenously instead of pethidine during anaesthesia.

Boulton, T. B. *Anaesthesia* 1955 10 33.

† Gold, M. I. and Stone, H. H. *Anesthesiology* 1957 18 357.

† Gravenstein, J. S. and others *New Engl. J. Med.* 1956 254 877.

respiration rate (ii) The lung volume i.e. the volume which dilutes each inspiration of gas or vapour (iii) The pulmonary blood flow which carries away anæsthetic gas from the alveoli so lowering its alveolar partial pressure (iv) The solubility of the gas or vapour in the blood. A soluble vapour e.g. ether is more easily carried away in the blood and so the alveolar tension of such a vapour will build up slowly (The partition co-efficient equals the ratio of the concentration of the gas in the blood to the concentration of that gas in air at equilibrium) (v) The partial pressure of the gas in the mixed venous blood coming back to the lungs since this brings gas back to the alveoli it helps to raise the alveolar tension.

- 2 The cerebral blood flow depends on (a) The cerebrovascular resistance which is influenced by the blood viscosity the intracranial pressure atheroma and tone of blood vessel walls. Vascular tone is influenced by (i) Chemical and (ii) Neurogenic factors. (i) Chemical inhalation of 5 per cent of carbon dioxide increases cerebral blood flow by 75 per cent while a low  $p\text{CO}_2$  reduces blood flow moderate hyperventilation reducing it 35 per cent likewise inhalation of 100 per cent oxygen decreases cerebral blood flow 12 per cent and inhalation of only 10 per cent of oxygen has a similar effect to 5 per cent carbon dioxide. (ii) Neurogenic factors are not very important as vasoconstrictor tone is seldom controlled by nervous impulses except possibly in acute lesions. Cerebral blood flow also depends on (b) The mean arterial blood pressure. In essential hypertension the cerebral blood flow is not proportional to the greatly raised arterial blood pressure. It also depends very considerably on (c) Gravity.

According to Guedel the agent reaching the tissue cells is directly influenced by —

- 1 The partial pressure of the agent inspired. Gases and vapours diffuse from a zone of high to a zone of low partial pressure the greater the difference the more rapid the diffusion.
- 2 The respiratory minute volume. Deep breathing increases shallow breathing decreases the available alveolar membrane exposed to the vapour.
- 3 The degree of permeability of the alveolar membrane. Mucus may interfere with absorption of vapour.
- 4 The time required for the whole volume of blood to pass through the lungs. This is important when nitrous oxide is the agent and is usually just over one minute.
- 5 The volume of blood supplied to the various organs. The brain receives the greatest fats the smallest volume.

**Balanced Anæsthesia** —Balanced anæsthesia really started in 1911 when George Washington Crile of Cleveland Ohio taught that psychic stimuli must be obliterated by light general anæsthesia while the noxious impulses due to surgery must be blocked by local analgesia—the so called theory of anoci association. In 1926 John S. Lundy of the May Clinic introduced the term

**Theory of Inhalation Anæsthesia continued**

there is inhibition of a carrier of energy which operates as an intermediate between the pyruvate of the cell and the cytochrome system. Cells of the central nervous system of most recent phylogenetic development are the most sensitive to oxygen lack.

- 2 That anæsthetic agents are readily absorbed by lipids hence brain cells are specially susceptible to their action (Meyer Overton 1899-1901). Partition of a drug between lipid and water phases reflects its biological potency as a narcotic. This is true for volatile anæsthetics but not for chloral hydrate or barbiturates which are both more hydrophilic than lipophilic.
- 3 That anæsthetics cause changes in cell metabolism of a physico-chemical nature e.g. precipitation of colloids (Claude Bernard 1875) changes in surface tension changes in permeability of cell membranes (Lillie 1918) changes in viscosity etc.
- 4 That anæsthetics act by changing electric polarity of cells of nervous system. Both ether and chloroform reduce the frequency and voltage of action potentials. The brain usually is electronegative to the rest of the organism but under anæsthesia it becomes more positive.

None of these theories is wholly satisfactory.

Thus available oxygen is reduced either by giving a subnormal amount of oxygen to the tissues or by giving an adequate amount of oxygen together with a tissue poison which prevents its utilization. The brain can be subjected to a degree of oxygen lack sufficient to cause anæsthesia at a time when other tissues are not seriously affected. Similarly as the brain receives a greater blood supply and contains more lipid in its cells than other tissues the amount of an anæsthetic agent absorbed by the brain is greater than that absorbed by other tissues. The ratio of the amount of a given anæsthetic absorbed by the lipid of the cell to the amount that leaves the cell in the venous blood is constant. This is the fat/water coefficient constant. The higher the value of this ratio the greater is the anæsthetic potency of the drug.

**MECHANISM OF INHALATION ANÆSTHESIA**

It depends on the laws of diffusion of gases and vapours. The tension of an inert gas or vapour in the brain depends on two primary factors (1) The tension of gas in the arterial blood and (2) The supply of that blood to the brain the cerebral blood flow.

- 1 The tension of gas in the arterial blood depends on (a) The tension of gas in the alveoli (b) The nature of the pulmonary diffusing surfaces which is influenced by the size of the lungs the thickness of the diffusion membranes the presence or absence of œdema and secretions and the pulmonary blood flow. In health diffusion is rarely a limiting factor and therefore the arterial tension of a gas usually equals its alveolar tension.

The alveolar tension depends on (i) The effective respiratory volume i.e. the tidal volume minus the dead space multiplied by the

*Advantages* are (a) Ability to control the blood carbon dioxide level (b) Even anæsthesia (c) Some conservation of heat and moisture from the patient. *Disadvantages* are (a) Cost of apparatus (b) Clumsiness and lack of portability (c) Cost of the gases (d) Carbon dioxide accumulation may cause hyperpnœa (e) Exhalation valve unless very loose will cause resistance to expiration

- 4 **Intermittent Closed Method.**—Popularized by Clover in 1877 whose inhaler was later modified by Hewitt. The face piece closely fits the patient's face and is attached to an ether chamber to the other end of which is fitted a breathing bag. The patient breathes into the bag and the mask is then firmly applied to the face. Ether vapour is gradually admitted to the mask being vaporized by the patient's breath. As carbon dioxide builds up hyperpnœa is produced. The face-piece must be lifted up every four or five breaths to allow inspiration of fresh air and escape of carbon dioxide. A tap allows oxygen or nitrous oxide to be added for induction. The method is seldom used to day and is only interesting from the historical viewpoint.
- 5 **Closed Method with Carbon Dioxide Absorption**—See Chapter VII
- 6 **The E.M.O. Ether Inhaler**—This has displaced the Oxford Vaporizer and is designed to deliver to the patient ether by his own respirations or by positive pressure from the Oxford Inflating Bellows any desired concentration of ether vapour irrespective of changes in the temperature of the liquid anæsthetic.
- 7 **The Endopharyngeal Insufflation Method.**—The introduction<sup>20</sup> into the naso- or oro-pharynx of an anæsthetic mixture by means of the side tube of a pharyngeal airway a catheter the breathing tube of a Boyle Davis gag or a mouth hook. Vaporization of the volatile agent is produced by air or gases passing over or through the liquid the motive force is either a pump or the decompression of a gas or gases released from a cylinder. It is useful in small children and in certain operations on the throat nose or ear in which endotracheal intubation is not used. The Junker bottle and Shipway's apparatus are suitable for this method of anæsthesia as are also the commonly used continuous flow machines.
- 8 **Draw over Method**—Air and if necessary oxygen are drawn over the surface of a volatile liquid or liquids. Inspiration of the patient is motive force. Vaporization may be helped by the use of a gauze wick. Marrett's apparatus (q.v.) is an example and so is the E.M.O. Inhaler and the inhalers designed for the self administration of trichlorethylene and air.

### THE STAGES OF ANÆSTHESIA FOLLOWING THE INHALATION OF GASES AND VAPOURS

Most anæsthetists now follow the plan of Guedel, who described the stages of anæsthesia and much else of value in his book *Inhalation Anæsthesia* (The Macmillan Co. London 1937). The signs mostly



**Balanced Anæsthesia continued**

balanced anæsthesia for a combination of agents such as pre medication regional analgesia and general anæsthesia with one or more agents so that pain relief was obtained by a nice balance of agents and techniques

**METHODS OF INHALATION ANÆSTHESIA**

Agents used for this may be liquid or gaseous at normal temperature and pressure. The former must be vaporized before administration.

**1 Open Method**—Introduced by Sir J. V. Simpson for use with chloroform in 1847. He used a folded handkerchief. To day the Schimmelbusch mask is employed which is a modification of Skinner's wire frame of 1862. Plenty of air can be inhaled through the gap between the mask and the face. Useful to day for giving chloroform ethyl chloride divinyl ether and occasionally trichlorethylene. Used by Lawson Tait of Birmingham from 1873 onwards it was popularized by Prince of Chicago for use with ether in 1895. Davis of the Mayo Clinic further popularized the method in the early 1900s.

**2 Semi open or Perhalation Method**—The mask is designed to fit the contour of the patient's face accurately. Examples are Ogston's mask and Bellamy Gardner's mask. A layer of gamgee between the mask and face further prevents air entry—other than through the gauze covering the mask. With this method some carbon dioxide build up occurs.

These methods do not deserve the disdain they usually receive. *Advantages* are (a) Immediate safety (b) Cheapness (c) Portability (d) Ease of administration (e) Minimal dead space. *Disadvantages* are (a) Uneven anæsthesia due to variations in concentration of vapour (b) Risk of fire (c) Wastefulness (d) Atmosphere of theatre becomes laden with vapour (e) Damage to eyes or skin of patient from anæsthetic liquid.

**3 Semi closed Method**—Gas and oxygen with or without the vapour of volatile agents are delivered to the patient via a closely fitting face piece, a breathing bag and an adjustable expiratory valve are present. During inspiration the patient inhales from the bag while part of his expiration passes out through the valve and part passes into the bag. An expiratory valve of minimal resistance adequate tidal exchange together with a total gas flow greater than the minute volume of the patient are the important factors in carbon dioxide elimination while the use of a non rebreathing valve prevents this altogether.

Machines delivering gases are of two types—

- ✓ **a Continuous flow** gases delivered continuously to the face piece. Examples Boyle's machine Heidbrink machine.
- ✓ **b Intermittent flow** gases only delivered during inspiration being automatically shut off during expiration if the delivering pressure is equal to atmospheric pressure. If it is increased the machine becomes a continuous flow type. Examples McKesson Walton machines.

**Fourth Stage—Respiratory Arrest**—Lasts from onset of complete respiratory paralysis to cardiac failure and death. All reflex activity is lost and pupils are widely dilated (except under cyclopropane with the patient fully oxygenated). Artificial respiration must be employed whenever breathing stops although active breathing may restart spontaneously. Death in the absence of artificial respiration depends on the amount of tissue oxygen and the ability of the myocardium to withstand anoxia.

Pfeiffer of the University of Illinois has very usefully divided the fourth stage into two planes. Plane I is the plane of respiratory paralysis with an intact circulation. Plane II is the plane of both respiratory and circulatory paralysis, i.e. of medullary paralysis. According to this classification the apnoea so easily produced using cyclopropane would be stage IV plane 1 while the apnoea associated with gross overdosage of e.g. chloroform would be stage IV plane 2.

### THE SIGNS OF ANÆSTHESIA FROM THE INHALATION OF GASES AND VAPOURS

Unconsciousness, abolition of reflexes, muscular atony and paralysis of respiration are due to depression respectively of the cerebral cortex, the mid brain, the spinal cord and the medulla (Fig. 2).

	RESPIRATION INTERCOSTAL DIAPHRAGM	OCULAR MOVEMENTS	PUPILS NO PREMEDICATION	EYE REFLEXES	SECRETION OF TEARS	LARYNGEAL AND PHARYNGEAL REFLEXES	RESP. RESPONSE TO SKIN INCISION	MUSCULAR TONE
STAGE 1								
STAGE 2								
STAGE 3								
PLANE II								
PLANE I								
STAGE 4								

Fig. 2—The levels of disappearance of reflexes (after Guedel)

- A. Respiration**—Inspiration is active depending on muscular contraction; expiration is normally passive, the accessory muscles only being used to overcome obstruction or in hyperpnea.

### The Stages of Anæsthesia *continued*

of disturbances of reflexes were worked out about 1920 using open ether and they do not appear so clear cut when some modern agents and techniques are used. An obstructed airway and hypoxia may alter the signs which are to be taken as a guide to and not as absolute criteria of depth of anæsthesia. The signs do not apply in the same way to cyclopropane or nitrous oxide while the use of relaxants masks many of them especially the respiratory signs.

### First Stage—Analgesia due to Depression of the Sensory Cortex

—An attempt has been made using ether to subdivide this stage into three planes in the deepest of which true analgesia is said to co exist with cerebation and co operation from the patient.\* Lasts from beginning of induction to loss of consciousness. The pain sense is progressively lost until it is absent when unconsciousness is reached. Mental control is present. Total analgesia is not easy to attain as onset of second stage with its restlessness is difficult to prevent. It is used to relieve the pains of labour, dental drilling and dressing of wounds etc. Nitrous oxide, trilene and chloroform are the drugs usually employed.

### Second Stage—Delirium due to Inhibition of Cortical and Subcortical Control Levels

—Lasts from onset of unconsciousness to onset of automatic breathing. There may be struggling or breath holding, vomiting or coughing. The inhibition from the higher cerebral centres is removed causing loss of self-control. External stimuli especially auditory—the last of the special senses to be abolished—may produce a violent reaction depending on the emotional state of the patient. Pupils may be dilated, but react to light. Delirium may appear during emergence from deep anæsthesia especially that due to cyclopropane. Sudden deaths—presumably from ventricular fibrillation—have occurred in this stage when chloroform has been used.

Adequate premedication, rapid smooth induction, quiet surroundings and reasonable restraint will usually minimize delirium. Induction with an adequate dose of intravenous barbiturate abolishes the second stage.

### Third Stage—Surgical Anæsthesia

—Lasts from the onset of regular breathing to apnoea from respiratory paralysis. It is the stage of progressive muscular depression and is separated into four planes—

PLANE 1—From onset of automatic respiration to cessation of eyeball movement.

PLANE 2—From cessation of eyeball movement to commencement of intercostal paralysis.

PLANE 3—From commencement to completion of intercostal paralysis.

PLANE 4—From onset of complete intercostal paralysis to diaphragmatic paralysis.

Morton emphasizes that the force of expiration is reduced with increasing depth of anaesthesia

In nervous patients rapid breathing may persist well into Plane 3 It must be distinguished from tachypnoea due to oxygen lack Slow breathing is usually due to overdose of sedative pre medication or of pethidine but this is not always so even if ether is the agent Trilene readily causes rapid breathing if given in too high a concentration

## B Eye Signs —

- 1 **EXTRINSIC MUSCLES** —Activity of the muscles of the eyeball is well marked in Stage I It is due to impulses coming from the superior colliculus to the pons and thence to the muscles by the third fourth and sixth nerves In Stage III Plane 1 it is progressively reduced until it is abolished at the bottom of this plane The activity may consist of oscillation squint or the eyes may be pulled up or down When Plane 2 is entered the eyeballs usually rest in the normal centre position In extreme hypoxia the eyeballs may be strongly pulled either upwards or downwards while the pupil is dilated
- 2 **PUPILS** —Opiates as premedication tend to produce miosis atropine and scopolamine in large doses mydriasis If both are given the opiate effect usually predominates In the first stage the pupils may be dilated reflexly due to emotion and psychosensory impulses in the second stage dilatation is due to sympathetic stimulation of the dilator muscle of the iris In Stage III Plane 1 the pupils return to normal and then progressively dilate until the maximum is reached in Plane 4 This is paralytic due to depression of the sphincter muscle by the anaesthetic agent This maximum is not total dilatation however which is only seen as the result of anoxia and the paralysis of the sphincter it causes

Horner's syndrome (small pupil exophthalmos and ptosis) may cause confusion It may follow cervical sympathetic paralysis associated with (1) Birth injury (2) Retropharyngeal abscess (3) Tuberculosis of the apex of a lung (4) Intra cranial lesions (5) Cervical rib etc

## NOTE ON ANATOMY OF NERVE SUPPLY OF IRIS —

- a *Parasympathetic* —The connector cells lie in the oculomotor nucleus in the floor of the aqueduct of Sylvius from which fibres pass with the oculomotor nerve to the ciliary ganglion From this post ganglionic (excitor) fibres pass in the short ciliary nerves to the constrictor muscle of the pupil and the ciliary muscle Function contraction of pupil and accommodation
- b *Sympathetic* —The connector cells lie in the lateral horn of the first and second thoracic segments pass out in the anterior root of T1 in the white ramus of T1 to the inferior cervical ganglion and ascend the sympathetic trunk to the superior cervical ganglion where excitor cells are located Post ganglionic fibres pass with the internal carotid artery into the skull and join the cavernous plexus

The Signs of Anæsthesia *continued*

**FIRST STAGE**—Respiratory volume gradually becomes increased due to stimulation of the respiratory centre by the anæsthetic (especially ether) Too strong a vapour or emotion may interfere with breathing

**SECOND STAGE**—Breathing may show many abnormalities but they are of no significance

**THIRD STAGE**—

**PLANE 1**—Onset of regular automatic breathing Depth of breathing depends on the relationship between the respiratory threshold to stimulation and the amount of stimulant—carbon dioxide—in the blood When ether is used there is usually some hyperpnœa which gets less after 15–20 minutes Premedication depresses while surgical trauma stimulates respiration in this and the two succeeding planes

**PLANE 2**—Breathing remains regular and deep not very different from Plane 1

**PLANE 3**—The plane of progressive intercostal paralysis first described by John Snow later by Paul Bert and again in 1899 by J Hughlings Jackson and James Collier Recognized as a sign of anæsthesia in 1924 by Albert Miller and placed in its correct position in Guedel's table by Ralph Waters Above Plane 3 intercostal and diaphragmatic breathing are synchronous the chest and the abdomen rising together With increasing intercostal paralysis the chest lags behind the abdomen a change often felt before it is seen Depth of respiration is reduced while the interval between inspiration and expiration increases at the expense of inspiration which becomes relatively shorter in duration Decreased depth of respiration may be made up for by increased rate Passive expiration does not change as much as active inspiration Increased diaphragmatic movement compensates for inactivity of the intercostals and may prove annoying to the abdominal surgeon especially as it is sometimes jerky Lightening of the level of anæsthesia will improve this Diaphragmatic paralysis preceding intercostal paralysis has been reported

**PLANE 4**—The thorax becomes stationary with complete intercostal paralysis There may be retraction of intercostal spaces during inspiration Tidal exchange may be inefficient so that oxygen lack and carbon dioxide excess may result Breathing is slow shallow and irregular When the thorax and abdomen move in opposite directions with complete intercostal paralysis respiration is termed external paradoxical respiration a similar appearance may accompany respiratory obstruction The term is not to be confused with internal paradoxical respiration in open pneumothorax

With emphysema increasing age and rigidity of the thoracic cage thoracic movements become less easy to assess

**FOURTH STAGE**—Marked by onset of apnœa During recovery the same changes take place in the opposite order

or by a relaxant. This is explained by Harris as being due to the forcible and unopposed action of the crura of the diaphragm transmitted to the central tendon the pericardium the lung roots and the trachea in the presence of an atonic state of the chondrosternal fibres of the diaphragm. It is seen in third and fourth plane anaesthesia and Guedel suggests that it may be associated with too much carbon dioxide in the blood.

- 7 CARINAL.—Stimulation of the carina as by a long endotracheal tube or bronchoscope may cause coughing anywhere above Stage 4.
- 8 ANAL SPHINCTER.—If this is rapidly stretched laryngospasm or hyperpnoea may occur if the patient is above Stage 4. In the days before artificial respiration was as frequently employed as it is now this reflex was used to stimulate breathing during periods of apnoea.
- 9 TRACTION REFLEXES.—Pulling on peritoneum mesentery liver etc. may cause hyperpnoea reflex contraction of the muscles of the anterior abdominal wall and laryngospasm. Or it may cause temporary apnoea. Abolished in Plane 4.

Harris\* slightly alters Guedel's classification. His four stages are —  
 Stage I The depression of the higher centres—corresponds to Guedel's classification Stages I II III Plane 1  
 Stage II The depression of sensory co-ordination—corresponds to Guedel's classification Stage III Plane 2  
 Stage III The depression of motor co-ordination—corresponds to Guedel's classification Stage III Planes 3 4  
 Stage IV The depression of the medulla—corresponds to Guedel's classification Stage IV  
 It consists of failure of the respiratory centre failure of the vasomotor centre and failure of the cardiac centre.

Laycock's four stages† are —

- I Consciousness with disorientation and analgesia
- II Unconsciousness with reflex activity
- III Unconsciousness with reflex depression
- IV Respiratory paralysis

The e newer classifications are useful when the patient has been anaesthetized with intravenous thiobarbiturates and muscle relaxants.

It has been suggested‡ that a word other than anaesthesia is needed to indicate the state of combined sensory mental and motor block and the word *nothria* (rhyming with *no fear*) has been recommended derived from it would be *nothrogen* (an agent producing nothria) *nothretize* to produce the state of nothria *nothretist* *nothrology* *nothrologist* etc. The state of nothria comprises four components (1) Sleep (2) Sensory block (3) Motor block (4) Suppression of reflexes.

Harris T. A. B. *The Mode of Action of Anaesthetics* 1951 ch. 15 Edinburgh E. & S. Livingstone.

† Laycock, J. D. *Anaesthesia* 1953 8 14.

‡ Woodbridge Philip D. *Anesthesiology* 1957 18 536.

*Eye Signs continued*

in the cavernous sinus Further course (1) In sympathetic root of ciliary ganglion and short ciliary nerves (2) To Casserian ganglia nasociliary nerves (1st division of 5th nerve) and long ciliary nerves to the eye

**3 OTHER EYE REFLEXES —**

- a EYELASH —Gently touching eyelashes causes contraction of the lids Reflex disappears on entering second stage
- b EYELID —Gently raising the upper lid causes contraction of the lids Reflex disappears on entering third stage
- c CONJUNCTIVAL —Gently touching palpebral conjunctiva causes blinking a reflex which disappears at bottom of Stage III Plane 1
- d CORNEAL —Gently touching cornea produces contraction of lids Reflex disappears in middle of Plane 2 The afferent pathway is the fifth nerve the efferent the seventh
- e LIGHT —Exposure to strong light causes the pupil to contract Reflex abolished in upper Plane 3
- f LACRIMATION —This is greater than normal in Planes 1 and 2 Thereafter it rapidly decreases until the dull glazed eye of deep anaesthesia results

**C Other Reflexes —**

- 1 SWALLOWING —Occurs at upper border of Plane 1 i.e. in the lightest 3rd stage anaesthesia Rapid deepening of anaesthesia at this point may avoid vomiting
- 2 VOMITING —Occurs at lower border of second stage i.e. in the deepest 2nd stage anaesthesia These signs are only of importance in ascent from deeper anaesthesia when swallowing may be a warning of impending vomiting Swallowing and vomiting mark the transition from the 3rd to the 2nd stage Vomiting may indicate oxygen lack when nitrous oxide is the agent
- 3 SKIN —Movement of limbs and deep breathing are the reflex responses to skin stimulation Abolished in Plane 2
- 4 POSTERIOR PHARYNGEAL —Stimulation of the mucosa of the throat by airways mucus or vomitus produces gagging or coughing Abolished at bottom of Plane 1 Afferent and efferent pathways the ninth and tenth nerves
- 5 LARYNGEAL —Coughing and adduction of the cords are produced by stimulation of the nerve-endings in the larynx and epiglottis Reflex abolished in upper Plane 2 Laryngeal spasm due to stimuli arising in the abdomen anus or cervix uteri etc (Brewer Luckhardt reflex) may not be abolished until Plane 4 Inflammation of upper respiratory tract increases its reflex irritability so that coughing may persist well into Plane 2 or even Plane 3 Thiopentone makes some patients cough an effect sometimes very difficult to quell
- 6 TRACHEAL TUG —This jerky depression of the thyroid cartilage synchronous with inspiration is often seen when the intercostal muscles are paralysed either by deep anaesthesia

about Rate of induction must be the quickest the patient will tolerate (exception chloroform) Inhalation anaesthesia is more often

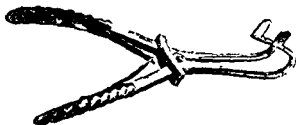


Fig 4—Ferguson's gag with Ackland jaws. (British Oxygen Gases Ltd.)

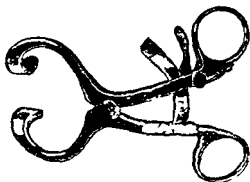


Fig 5—Doyen's gag (British Oxygen Gases Ltd.)



Fig 6—Guedel's pharyngeal airway (A. Charles King Ltd.)



Fig 7—Waters' pharyngeal airway (A. Charles King Ltd.)

used for maintaining anaesthesia than for inducing it intravenous thiobarbiturates are the usual means of induction



### LEVELS OF ANÆSTHESIA FOR VARIOUS OPERATIONS

Surgeons differ markedly in their requirements and no hard and fast rules can be made

The following procedures can be done in relatively light planes of anæsthesia and they do not require muscular relaxation operations on thyroid breast brain mastoid skin grafts

Abdominal surgery demands adequate muscular relaxation and quiet breathing It is more important to produce these than to strive for any predetermined level of anæsthesia Gastrectomy in a powerful young man will require greater depth than appendicectomy in a thin young girl Lower second or upper third plane is usually necessary for laparotomies

While light anæsthesia is desirable the level must not be allowed to become too light Reflexes arising from the operation site and from the patient's airway must be suppressed The combination of a strong stimulus with high reflex irritability may result in cardiac inhibition and sudden deaths have been reported following such procedures as endotracheal intubation cervical dilatation etc

Providing the surgeon is satisfied the lighter the level of anæsthesia the better but powerful motor responses to sensory stimuli should always be abolished and tranquility is always desirable especially so in poor risk patients

If an endotracheal tube is in position the level of anæsthesia must be kept below Plane 1 otherwise coughing may result if inhalation anæsthesia alone is used In long procedures the laryngeal reflex may eventually become fatigued and this is helped by topical analgesia applied to the laryngeal mucosa

### ADMINISTRATION OF INHALATION ANÆSTHETICS

The following should be at hand before the induction of every general anæsthetic (1) Mouth prop (2) Gag (Figs 3-5) (3) Gauze or tongue forceps to draw tongue forwards (4) Pharyngeal airway (Figs 6-8) (5) Nasopharyngeal tube (6) Face mask (7) Laryngoscope (8) Endotracheal tubes



Fig 3—Mason & Co (British Oxygen Gases Ltd.)

The patient's identity and diagnosis must be established and written permission for operation must be seen

False teeth must be removed dose and time of premedication must be checked presence or absence of stomach contents must be inquired

- a Spasm of Jaw Muscles* — Remedy is to push lower jaw forward by pressure behind the angles of the mandible. Occasionally elevation of the point of the jaw is sufficient. If obstruction is not thus relieved a well lubricated nasopharyngeal airway should be inserted into a nostril and manipulated so that its distal end lies just above the glottis. A safety pin prevents it going too far down. The distance between nares and epiglottis equals that between nares and tragus of ear. Calibre of tube should be that of Magill endotracheal tubes sizes 7-10. With the tube in place and obstruction relieved anaesthesia is deepened until the jaw can be easily opened for the insertion of a pharyngeal airway. A nasopharyngeal airway is tolerated at a lighter plane of anaesthesia than is a pharyngeal airway.

To overcome jaw spasm it was sometimes necessary to prise the jaw open with a gag, draw the tongue forward and insert a pharyngeal airway. The jaws of Ackland's gag are well adapted for this manoeuvre. Care must be taken of the teeth. The introduction of short acting muscle relaxants should enable the tightly clenched jaw to be opened atraumatically.

- b Flaccidity of the Jaw Muscles* — Remedy is to manipulate the jaw and to insert a pharyngeal airway (e.g. Phillips Guedel or Waters) the chief function of which is to keep the tongue away from the pharyngeal wall.

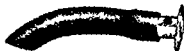
- 3 OBSTRUCTION ABOVE THE GLOTTIS — This may be due to a swab, a tooth, a foreign body, saliva, vomitus, blood or oedema. The obstructing material must be removed by the fingers, gravity, swabbing or suction. Very rarely dislocation of the epiglottis or cysts or tumours of the epiglottis are encountered. To relieve these cases lower the head of the table, open the jaw, withdraw the tongue, apply suction and administer oxygen. If unsuccessful first feel the epiglottic region and then look at it through a laryngoscope. Intubation may be necessary. The last remedy is tracheotomy and it is extremely rarely necessary.

- 4 OBSTRUCTION AT THE GLOTTIS — This is due to (a) Impaction of the epiglottis into the larynx due to its abnormal overhanging shape, gravity and muscular relaxation. The force of the inspiratory stream tends to maintain impaction while positive pressure inflation will not overcome the obstruction. The remedy is visual disimpaction via a laryngoscope after ensuring proper relaxation. (b) Sphincter like closure of the aryepiglottic folds allowing expiration but impeding inspiration. (c) Approximation of the ventricular ligaments or false cords. (d) Spastic closure of the rima glottidis by the vocal cords. Their dome shaped inferior surfaces allow escape of air but their level superior surfaces prevent its entry. It may result from stimulation of the mucosa of the larynx or from stimuli arising at the site of operation (Brewer Luckhardt reflexes) or from a combination of these factors with too light a plane of anaesthesia. It is usually worse on inspiration than

Administration of Inhalation Anæsthetics *continued***Difficulties —**

Delirium of the second stage must not be allowed to interfere with induction the patient must be physically controlled by a nurse or porter

Swallowing gagging breath holding coughing and laryngeal spasm may be due to emotion or to the use of too strong a vapour In the latter case vapour concentration must be decreased and later increased Addition of a little carbon dioxide will also help



*Fig. 8 — Phillips's pharyngeal airway (Bristol Oxygen Gases Ltd.)*

**RESPIRATORY OBSTRUCTION** — The signs are (1) Inadequate tidal exchange suggested by the small excursion of the reservoir bag or the weakness of the sounds of breathing (2) Retraction of the chest wall and of the supraclavicular infraclavicular and suprasternal spaces (3) Excessive abdominal movement due to an overworking diaphragm (4) Use of accessory muscles of respiration (5) Noisy breathing (unless obstruction is absolute and complete) (6) Cyanosis (7) The natural heave of the chest and abdomen becomes replaced by an indrawing of the upper chest and an outpushing of the abdomen because of strong diaphragmatic action the lower thoracic region is almost at rest This is the see saw movement of complete respiratory obstruction and is one type of external paradoxical respiration In partial cases it is not so marked Respiratory obstruction is the cause of many deaths occurring in the operating theatre as well as in patients suffering from strokes drug depression coma and head injury It is evil in three ways —

- a It interferes with gaseous exchange
- b It calls for increased effort on the part of the patient  
The increased negative pressure during inspiration may result in pulmonary œdema
- c It interferes with adequate anæsthesia and retards induction

It must be remedied in all cases after its cause has been diagnosed It may be due to —

- 1 **OBSTRUCTION AT THE LIPS** — Especially in edentulous patients A small mouth prop (e.g. Baker's airway London Hospital prop) is the cure
- 2 **OBSTRUCTION BY THE TONGUE** — Due to approximation of the tongue to the posterior pharyngeal wall (swallowing the tongue) It may be especially dangerous between the time that the patient leaves the hands of the anæsthetist and the return of his reflexes and muscle tone It is very much less likely to occur if the patient is lifted into the semi prone or tonsil position May be accompanied by —

after a relaxant has been injected. If laryngeal spasm does not pass off and progressive hypoxia is in evidence artificial respiration must be started at once if an endotracheal tube cannot be inserted —

- a Apply pressure to reservoir bag filled with oxygen after closing the expiratory valve and holding mask to face or—
- b Blow into face mask or—
- c Compress the lower chest and then draw the costal margins upwards and outwards

In any of these methods an oropharyngeal or nasopharyngeal airway may be required

If patient is in *extremis* a large needle can be inserted into the larynx through the cricothyroid membrane and air can be injected from a 20 ml syringe repeatedly or half a litre of oxygen can be insufflated. Cocaine can also be squirted on to the under surface of the cords from a needle inserted through the cricothyroid membrane. Tracheotomy is needed only on the rarest occasions

- 5 LOWER RESPIRATORY OBSTRUCTION — This takes place in the bronchioles and is of great importance. It is upon the patency of the bronchiolar lumen and the quiescence of the bronchial reflexes that smooth anaesthesia largely depends (Nosworthy)\*. Bronchial and bronchiolar reflexes result from too light a plane of anaesthesia and this may be associated with the stimulation of sputum, artificial airways, anaesthetic vapour, movement of the patient and also from stimuli arising in the area of operation. The response is great or small depending on the reflex irritability of the patient, his threshold of irritability. This is raised in old and ill and lowered in nervous and young patients and those in severe pain. The sensitivity of the respiratory tract increases from above downwards being greatest at the carina. Acute infection increases sensitivity as also does tuberculosis. Bronchiectasis does not increase sensitivity to the same degree. A tendency to asthma strongly predisposes to bronchial spasm and wheezing.

Anaesthesia is incomplete unless the threshold of irritability has been raised sufficiently to prevent the stimulus of the moment from breaking through.

#### OTHER RESPIRATORY ABNORMALITIES —

- 1 CE SATIO : OF BREATHING — May be associated with (a) Respiratory obstruction, abortive attempts at breathing can usually be seen. (b) Depression of the respiratory centre by sedatives, general anaesthetics etc. Easily caused by thiopentone and cyclopropane. (c) Apnoea following hyperventilation. (d) Depression of the Hering Breuer reflexes during intermittent positive pressure respiration. (e) Reflexes resulting from stimulation of the coeliac plexus and its offshoots.

Administration Difficulties—Obstruction at the Glottis *continued*

expiration and causes a high pitched tenor sound. It is not influenced by altering the hold on the jaw unlike the lower pitched noise of pharyngeal obstruction. (e) Occasionally in patients who have had a large dose of relaxant and have been intubated respiratory obstruction develops after extubation because the relaxed cords instead of becoming abducted during inspiration become sucked in by the air-stream.

Local stimulation may be due to a vapour too concentrated blood or mucus or to irritation from an airway temporarily reduce vapour strength and partially withdraw airway suck out blood or mucus.

Occasionally in operations on the neck trauma to the recurrent laryngeal nerve causes spasm. Division of a nerve causes fixation of the cord midway between abduction and adduction. *Difficulty only occurs during hyperpnœa*.

Peripheral stimulation causing partial or complete spasm suggests the need for deeper anæsthesia. Such stimuli occur when the cervix uteri or anal sphincter is stretched when the celiac plexus or its branches are stretched when periosteum is separated from rib etc. Addition of carbon dioxide and pressure on the breathing bag during inspiration will increase speed of induction and help separation of the cords. Temporary cessation of surgical stimuli may occasionally be necessary. The parasympathetic stimulants thiopentone and to a less extent cyclopropane favour the activity of this reflex especially if an artificial airway is inserted when the plane of anæsthesia is light.

If a volatile anæsthetic is being given the vapour strength should be reduced and a little carbon dioxide given to increase the urge to breathe after which the vapour strength is progressively increased. If the patient is under the influence of an intravenous thiobarbiturate it may be necessary to ask the surgeon to interrupt his stimulating manœuvres for a few moments the condition can cause considerable difficulty and may call for more active treatment. If this is considered necessary one of the following can be given. (a) A short acting relaxant. (b) Trasentin, known also as adiphenine and as diphenyl acetyl-diethylaminoethanol hydrochloride. It is an antispasmodic and smooth muscle relaxant having a papaverine like effect on smooth muscle and an atropine like effect on parasympathetic nerve-endings. Has no drying effect on salivary glands. It acts quickly when injected intravenously in doses of 25–50 mg average 35 mg. Used also as an antispasmodic in diseases of intestinal renal and biliary tracts. It is slowly hydrolysed into diphenylacetic acid and diethylaminoethanol.

The passage of an endotracheal tube between the cords completely abolishes the condition and is made easier

intravenous injection of (a) procaine, (b) pethidine (c) aminophylline (250 mg repeated if required) (d) adrenaline or noradrenaline drip (1:250 000) (e) Suxamethonium intravenously. May occur in asthmatics. Bronchospasm may well be due to inadequate suppression of reflexes.

- ✓ 8 **TRACHEAL TUG**—This jerky type of inspiration is seen when the intercostal muscles and the sternocostal parts of the diaphragm are paralysed either by deep general anaesthesia or by muscle relaxants (T A B Harris). It may be due to the unopposed action of the crura pulling on the dome of the diaphragm and thence on the pericardium lung roots and tracheobronchial tree during each inspiration. The degree of tug depends on the depth of respiration and so it is more marked if ether is given than if cyclopropane is used. It disappears as soon as the intercostal muscles and the sternocostal parts of the diaphragm regain their tone and is a useful guide to the degree of muscular relaxation especially at the end of an operation.
- 9 **APNEUSTIC BREATHING**—As anaesthesia deepens the jerky type of breathing with tracheal tug may give place to a series of slow deep inspirations each one held for 30–90 seconds after which the air is suddenly expelled by the elastic recoil of the lungs. This may finally give place to gasping breathing and apnoea.
- 10 **HICCUP**—This may be seen especially when thiopentone gas and oxygen and a relaxant are used. It may be associated with too light a plane of anaesthesia nervousness of the patient (i.e. high reflex irritability) stimulation of vagal nerve-endings (e.g. during gastrectomy) by a raised blood carbon dioxide content. The following have been recommended as remedies: (a) Inhalation of amyl nitrite introduced into the circuit. (b) A short blast of strong ether vapour (Gray). (c) Inhalation of ammonia. The success of these three methods suggests that a reflex mechanism is involved as hiccup stops too rapidly to be due to absorption of the drug. (d) Addition of more relaxant e.g. a paralysing dose of suxamethonium. (e) Intravenous injection of methicidine. (f) Intravenous injection of pethidine. (g) Procaine block of the vagi at the oesophageal hiatus. (h) Vigorous hyperventilation via the reservoir bag. (i) Pressure on the eyeballs. After successful treatment the condition may recur later during the operation. The cause of this troublesome complication is not fully understood.
- 11 **CHEYNE STOKES BREATHING**—This may be seen after over dosage with respiratory depressant drugs e.g. after chlorpromazine. It also occurs in heart failure head injury and more rarely in kidney disease. When not due to drugs it has a bad prognostic significance. Can be temporarily abolished by the intravenous injection of 250 mg of aminophylline and sometimes by inhalation of oxygen with or without carbon dioxide. Biot's breathing is irregular periods of apnoea and hyperpnoea.

Administration Difficulties—Respiratory Abnormalities *continued*

e.g. traction on the gall bladder (f) Blood pressure raising drugs e.g. adrenaline (action on aortic and carotid baroreceptors) (g) Depression of aortic and carotid body (chemoreceptor) reflexes by a high oxygen tension following a period of hypoxia (h) Bronchoconstriction due to irritation of the upper respiratory tract parasympathetic stimulating drugs (e.g. thiopentone cyclopropane) light anaesthesia and surgical stimuli (i) Raised intracranial pressure (j) The use of muscle relaxants

- 2 **RAPID BREATHING**—This may occur in feverish patients and may also be due to respiratory obstruction. May follow oxygen lack or carbon dioxide accumulation. Frequent when too much trilene is given. Low blood pressure by failing to stimulate aortic and carotid sinuses may cause rapid breathing. An increased venous return causing a rise in pressure in the right auricle stimulates receptors there and causes tachypnoea (Harrison and Marsh reflex).
- 3 **SLOW BREATHING**—May be caused by sedatives given before operation and by pethidine or morphine given intravenously during operation. Bilateral cervical vagotomy also causes slow breathing.
- 4 **HYPERPNOEA**—May follow severe surgical stimuli e.g. abdominal traction reflexes. Otherwise it is likely to be due to respiratory obstruction hypoxia or carbon dioxide excess.
- 5 **HYPOPNOEA**—Shallow breathing may be due to —
  - a Deep i.e. 3rd or 4th plane anaesthesia
  - b Cyclopropane
  - c Intravenous barbiturates used for induction of anaesthesia
  - d Morphine or pethidine overdosage
  - e Myoneural block. During underventilation the molecular weight of carbon dioxide being 44 may cause it to diffuse less rapidly than oxygen which has a molecular weight of 32 so the raised blood-carbon dioxide is likely to be more marked than the low blood oxygen level. This respiratory acidosis may give rise to an increase in the blood adrenaline and noradrenaline levels.
- 6 **IRREGULAR BREATHING**—In deep anaesthesia shows overdosage and heralds respiratory arrest. In light anaesthesia may indicate ascent into second stage.
- 7 **BRONCHOSPASM**—In addition to spasm of the bronchial muscle there is often an oedema a type of urticaria of the bronchial mucosa and sometimes an oedema due to engorgement of the bronchial vessels. It is characterized by lower respiratory tract obstruction with (a) small tidal exchange (b) prolonged forceful expiration sometimes producing bucking movements (c) wheezing.

Caused by vagal stimulation either central e.g. cyclopropane thiopentone or peripheral e.g. by endotracheal tube or pharyngeal airway or the surgical stimulus. Should be treated by the addition of a little trilene or ether or by

stage of coughing or laryngospasm. The method needs experience especially when robust individuals are being anaesthetized and is surely very rarely employed to-day.

1 thyl chloride can be preceded in adults and in older children by 0.25-0.5 g of intravenous barbiturate which produces unconsciousness very pleasantly.

Induction with chloroform or chloroform ether mixtures is seldom justified as it is during the induction period that chloroform is especially dangerous.

Interbalational or semi-open methods increase speed of induction.

#### 15 SEMI CLOSED ETHER —

1 CONTINUOUS FLOW MACHINES (e.g. Boyle) — Gases are delivered from cylinders and their rate is measured on flow meters. Gases can if desired be directed over the surface of or bubbled through anaesthetic liquid contained in two bottles. Wide bore corrugated tubing connects the machine with the face piece while near the machine is a reservoir bag and near the mask an expiratory valve (the Magill attachment). A proportion of the expired gas is returned to the reservoir bag and rebreathed unless a non rebreathing valve is employed (See Figs 18, 19). The following factors may affect the concentration of vapour from volatile agents inhaled by the patient: (a) Distance from the bottom of the plunger to the surface of the agent. (b) The tap setting. (c) The volume of gas flowing. (d) The degree of eccentricity of the plunger and U tube (this can be avoided by soldering three pieces of copper wire on to the U tube†). (e) The temperature of the agent. (f) The volume of agent in the vaporizing bottle. (g) the type of anaesthetic system connected to the vaporizing unit. (h) The type of rubber used in the system. (i) The type of breathing. (j) The time from the commencement of vaporization.

Nitrous oxide is used to produce unconsciousness. After seeing that the cylinders are correctly coupled up a flow of 8-10 litres per minute of the gas is turned on and the face piece held a couple of inches over the patient's face. When consciousness is lost (abolition of eyelash reflex and onset of slight cyanosis) the flow is reduced to 6 litres a minute the mask is applied to the face while the ether tap is turned to its minimal vaporizing position which varies from machine to machine. Two litres a minute of oxygen are now added and the ether tap progressively turned until it is full on and then the plunger is depressed as fast as the patient will tolerate the increase in vapour strength. Addition of a half litre a minute of carbon dioxide will speed up induction. It is advisable that the total flow of gases should not be less than the minute volume i.e. 6 litres a minute and the oxygen percentage 5 or more if patient is to be sure of re-ceiving in his lungs at least

Mapleson W. V. *Brit J Anaesth* 1957 29 3  
† Hildreth J. *Ibid* 1957 29 3



**Administration Difficulties—Respiratory Abnormalities continued**

Obstruction to respiration may be due to some fault in the anæsthetic apparatus such as —

- 1 Kinking of the endotracheal tube or breathing tube
- 2 Obstruction of the endotracheal tube or breathing tube
- 3 Absence of gas flow to patient due to empty cylinders faulty flowmeters or reducing valve gauges

**Technique —**

**A OPEN ETHER**—Atropine or scopolamine should be the pre medication given intravenously just before the induction if necessary. If ether alone is used—an uncomfortable method—twenty or thirty minutes may be required to get the patient fully anæsthetized as a rapid increase in vapour strength may cause coughing and breath holding.

The mask is covered with fifteen thicknesses of gauze and as induction proceeds a gamgee pad with hole for nose and mouth can be allowed to separate the mask from the face cutting down air leaks (technically semi-open or perhalation). Ether is dropped on to mask as quickly as patient will tolerate it and during first quarter hour of surgical anæsthesia rate will approximate a hundred drops a minute. In second quarter hour this rate will be halved. If the patient objects to the vapour it is diluted with air by raising the mask. A gentle flow of carbon dioxide under the mask will hasten induction. When neck muscles relax head is turned to one side while the jaw is supported to procure a good airway. Full rotation of head with slight extension of neck is frequently all that is required to ensure a good airway. If necessary a pharyngeal airway can be introduced. The maximum concentration of ether under an open mask is about 15 per cent. With open ether there is always a reduction in the oxygen tension under the mask so 500–1000 ml of oxygen per minute should be added in every case. Reduction of oxygen tension under the mask falls on average from 150 mm Hg to 105 mm Hg and is due to displacement of atmospheric oxygen by carbon dioxide and by ether vapour which averages during induction 37–55 mm Hg partial pressure.

A slow progressive increase in concentration and a good airway are the secrets of a successful induction. As the anæsthetic state becomes established a given plane is maintained with the addition of less and less anæsthetic—the so-called law of diminishing resistance of Gull. This is because concentration in the blood comes to equal the concentration in the tissues.

Induction with *ethyl chloride* is quicker than with ether alone. It is gently sprayed on to a mask the concentration being increased as quickly as possible. The patient's normal breathing first becomes irregular then automatic and finally stertorous. Shortly after this point the mask is removed and a second one well soaked with ether is applied to the face so that the patient's deep inspirations draw in sufficient ether vapour to maintain surgical anæsthesia without an intervening

- L. THE CLOSED CIRCUIT WITH CARBON DIOXIDE ABSORPTION** (See also Chapter VII).—If ether is to be used induction is often quicker when a semi-closed circuit is used to start with. When the required level of anaesthesia has been reached the bag is filled with the mixture of gases being breathed the gases are shut off the expiratory valve is closed and the mask strapped firmly to the patient's face. A basal oxygen flow of 250–400 c.c. per minute is turned on and regulated so that the volume of gas in the bag remains constant. The soda lime is brought into

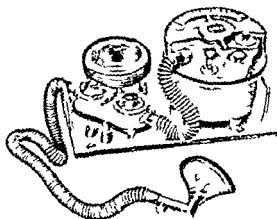


Fig. 1. McC. (The McC. Instrument Co. Ltd. Oxford).

circuit. Increased depth is obtained by carrying the total gases over the ether vaporizer as in the McKesson and Coxeter-Mushin apparatus or by bubbling the basal oxygen through both the bottles full of ether in the Boyle machine. In the last machine alternatively the circuit can be opened temporarily and a large volume of gas can be bubbled through ether to deepen the anaesthesia.

In addition to ether chloroform and vinylene can be used in a closed circuit but trilene should not be used in conjunction with soda lime.

- F. ASSISTED BREATHING** (Augmented supplemented complemented reinforced breathing intermittent positive pressure respiration (IIPR)).—This consists of increasing the tidal exchange of successive or of alternate breaths by manual pressure on the reservoir bag coincident with inspiration. Expiration takes place by the natural elastic recoil of the lungs. Escape of too much gas through the expiratory valve is prevented either by (1) closing it completely during inflation (e.g. the Salt

**Administration Technique—Semi closed Ether continued**

70 per cent of oxygen. It may be necessary in resistant patients to use both bottles with ether first the smaller one and then the larger. Maintenance of anæsthesia can often be smoothly carried along with the gases passing over the surface of ether in one bottle. If the tension of the spring of the expiratory valve is relatively high the mean pressure in the anæsthetic circuit and in the patient's respiratory tract will be increased and so will the effort required to expire through the valve. The main factors governing rebreathing are not the setting of the expiratory valve but the relationship between the respiratory minute volume of the patient and the flow of fresh gases into the circuit. The tension on the valve should therefore always be as light as possible.

It is becoming customary to follow nitrous oxide with a little trilene and then to add ether. Trilene is less irritating to the respiratory mucosa than ether and thereby a smoother induction is easier.

2. **INTERMITTENT FLOW MACHINES** (e.g. McKesson)—Gas mixtures at predetermined pressures and of predetermined percentage are delivered to the patient only when he inspires. The gases can be diverted over an ether or trilene vaporizing bottle. The expiratory valve is opened, the absorber indicator set at change, the nitrous oxide at 100 per cent and the pressure at 3 to 5 mm Hg. The mask is put on the patient's face and he inhales 100 per cent nitrous oxide until consciousness is lost when the ether pointer is turned to its minimal vaporizing position and 70 per cent oxygen set on the percentage dial. Ether or trilene concentration is increased as rapidly as possible and here too the addition of 300-500 c.c. of carbon dioxide a minute will hurry the induction along. (See Fig. 26.)

3. **THE EMO INHALER\*** (Fig. 9)—This has replaced the time honoured and very useful Oxford Vaporizer and like it was developed in the Nuffield Department of Anæsthetics in the University of Oxford. It will deliver a predetermined concentration of ether vapour in air irrespective of changes in the temperature of the liquid anæsthetic and is controlled by an automatic thermocompensator mechanism. A water compartment acts as a heat buffer. It is usually employed as a draw over apparatus but if combined with an Oxford Inflating Bellows can be used for intermittent positive pressure respiration. Rebreathing is prevented by the use of unidirectional valves †.

4. **THE EDISON ETHERISER ‡**—This utilises exothermic heat of wetting and adsorption of liquid and vaporizes ether on activated charcoal. The ether-air mixture resulting is a true gas in a state of superheat.

*Peetia & Instrument Co. Ltd. Oxford*

† Epsl in H.C. and McIntosh R.R. *Anæsthesia* 956 11 83  
Br. J. D.R. *Curr. Res. & Asth.* 1953 37 4 17

PROPERTIES OF SOME INHALATION ANESTHETICS

ANESTHETIC	MOLECULAR WEIGHT	BOILING POINT (°C.)	VAPOR DENSITY	LIQUID DENSITY	SOLUBILITY IN 100 PARTS OF WATER (mL.)	OIL/WATER SOLUBILITY	LIMITS OF FLAMMABILITY (per cent) $a = \text{in air } b = \text{in } O_2$	VAPOR CONCENTRATION FOR ANESTHESIA (vol./per cent)	BLOOD CONCENTRATION FOR ANESTHESIA (mg./per cent)
Diethyl Ether $C_4H_{10}O$	74	35	6	0.7	7.5	3.2	$a \pm 8.5$ $b \pm 8.2$	3.20	50-150
Diethyl Ether $C_4H_{10}O$	74	35	6	0.7	7.5	3.2	$a \pm 8.5$ $b \pm 8.2$	3.20	50-150
Diethyl Ether $C_4H_{10}O$	74	35	6	0.7	7.5	3.2	$a \pm 8.5$ $b \pm 8.2$	3.20	50-150
Cyclopropane $C_3H_6$	42	-34	1.4	—	33	34	$a \pm 4$ $b \pm 4$	7.23	2.5-17
Trichloroethylene $C_2Cl_3$	131	87.5	4.5	1.47	—	—	$a$ nonflammable $b \pm 10-64$	0.22	6-12
Chloroform $CHCl_3$	119	61	4.1	1.49	0.8	100	Nonflammable	0.52	25-70
Ethyl Chloride $C_2H_5Cl$	64.5	12	2.2	0.9	0.37	—	$a \pm 4$ $b \pm 4$	—	20-30
Ethyl Vinyl Ether $C_4H_8O$	72	35.8	2.5	0.6	0.8	45	$b \pm 2$	—	0-30
Nitrous Oxide $N_2O$	44	-18.1	1.5	1	150	32	Nonflammable	50-80	—
Halothane $CF_3CHClBr$	197	50	—	1.86	0.34	350	Nonflammable in oxygen 0.5-30	0.52	—
Fluoromethane $CF_3CH_2O-CH_2-CH_3$	126	42	4.4	1.13	0.4	94	$a \pm 4$ $b \pm 4$	3.8	17-38

Administration Technique—Assisted Breathing *continued*

and McHesson valves) or by partially screwing it down (C. J. Coxeter Heidbrink). Even better is the hole in the wall—an aperture in the wall of some convenient part of the breathing tube which can be closed by the finger during inflation. Such a device enables the pressure in the air passages of the patient to be returned to the atmospheric level suddenly and this in turn favours venous return to the heart. The total gas flow must be in excess of the minute volume of the patient—at least 8 litres per minute. Manually assisted breathing should be used frequently as all anaesthetic agents except nitrous oxide and ethylene together with pre-operative sedatives are respiratory depressants and raise the threshold of the respiratory centre to its natural stimulus carbon dioxide. There is thus a tendency for carbon dioxide to accumulate and for respiratory acidosis to be caused. By manually assisted breathing the tidal exchange should be kept equal to or slightly above normal. There is no evidence that respiratory alkalosis is harmful. Similarly assisted breathing removes the risk of hypoxia—provided that the inspired mixture contains an adequate tension of this gas.

Controlled breathing (qv) differs from assisted breathing in that in the former the respiratory mechanism is temporarily totally inactive.

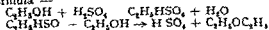
## CHAPTER VI

## AGENTS FOR INHALATION ANAESTHESIA

ETHER [ $\text{CH}_3\text{CH}_2\text{---O---CH}_2\text{---CH}_3$ ]

It is reported originally in 1540 by Valerius Cordus who called it sweet oil of vitriol. Used clinically for anaesthesia by W. E. Clarke and Crawford Long (1815-93) in 1842 but he did not publish his results. Introduced to the profession by W. T. G. Morton of Boston (1819-68) on Oct. 10, 1846. It did not attain much popularity in Britain until B. Joy Jeffries came from the U.S. with the American towel cone method of forcible induction in 1872. Two years later Clover introduced his gas—ether sequence. Before this time chloroform had been used almost as a routine. Open ether was reintroduced by Erice in America in 1895. It is still the safest all purpose anaesthetic. I hold it therefore to be almost impossible that a death from this agent [ether] can occur in the hands of a medical man who is applying it with ordinary intelligence and attention. (John Snow *On Chloroform and Other Anaesthetics* 1858)

**Manufacture**—By heating together concentrated sulphuric acid and ethyl alcohol which thereby becomes dehydrated according to the formula—



centre. There is peripheral vasodilatation especially of face and head—the effect of this on the meningeal and cerebral vessels is to raise the cerebrospinal fluid pressure. In deeper planes the vasomotor centre is paralysed so that peripheral vasodilatation is another cause of reduced blood pressure.

The cardiac output is increased at light planes, decreased at deep planes of anaesthesia. To produce cardiac standstill the ether concentration must be thrice that required to produce paralysis of the respiratory centre. This is in marked contrast to chloroform which is twenty-five times more toxic to the heart than ether.

Auricular extrasystoles may occur in light anaesthesia but are without significance. Adrenaline is compatible with ether.

Recent work\* would tend on the other hand to show that cardiac dilatation is obtained even with subanaesthetic levels of blood ether increasing progressively as the level of blood-ether rises. Clinically however experience would tend to regard these changes as relatively harmless.

The blood shows a reduction in plasma volume and an increase in viscosity due to contraction of the spleen (sympathetic effect) causing an increase in circulating red cells and haemoglobin. The white-cell count is also increased, the polymorphs by as much as 200–300 per cent, the increase lasting several days.

**RESPIRATORY SYSTEM**—Respiratory movements first increase due to mildly stimulating effects of ether vapour on the tracheo-bronchial mucosa and stimulation of the respiratory centre. They later decrease as anaesthesia deepens but do not become inadequate until Stage 3 Plane IV. Respiratory rate increases while amplitude decreases as anaesthesia deepens and the respiratory rate may be greater than thirty a minute during maintenance. In light surgical anaesthesia with ether the respiratory centre continues to respond to carbon dioxide and the blood tensions of this gas and of oxygen remain within fairly normal limits. This is not so in deep planes. Salivary but not bronchial secretions are increased while bronchial muscles are relaxed. Bronchial and upper respiratory cilia not inhibited by ether vapour. Vapour is irritating producing cough and laryngeal spasm and perhaps reflex apnoea if introduced too rapidly. Hence induction of anaesthesia should be gradual starting with a low ether tension. The Hering-Breuer reflex is depressed in Stage 3 Plane II but in Plane I the reflex inhibition of inspiration on increasing intrabronchial pressure lasts five to ten seconds. Useful in patients with tendency to bronchospasm and emphysema.

**CENTRAL NERVOUS SYSTEM**—Induces analgesia followed by excitement and then anaesthesia. Cerebral cell metabolism is depressed in descending phylogenetic order. Medullary depression is late and precedes serious cardiac depression. Meningeal and cerebral vessels dilate—an undesirable state in

**Ether—Manufacture continued**

The concentrated sulphuric acid is mixed with 95 per cent alcohol and heated to 140 C in a still and alcohol vapour passed continuously into the mixture. The ensuing vapour a mixture of ether alcohol and water is scrubbed with sodium hydroxide to remove the sulphur dioxide and passed through a fractionating column from the top of which ether and alcohol come off as vapour. This is treated with alkaline permanganate dried over calcium chloride and distilled.

**Physical Properties**—Colourless volatile liquid of molecular weight 74 and specific gravity 0.718. Boiling point 35 C specific gravity of vapour 2.6 (i.e. it is two-and-a-half times heavier than air). Oil/water solubility ratio 3.2 as 100 c.c. of oil dissolves 5000 c.c. of ether while 100 c.c. of water dissolves 1546 c.c. of ether. Ether vapour is highly inflammable and explosive in air between 1.83 per cent and 36.5 per cent. Explosive in oxygen between 2 per cent and 82 per cent. Ether vapour when it arrives at the patient's end of the corrugated tube is at or very near room temperature whatever its temperature on entering the tube. It is difficult to exceed 13 per cent concentration using an open mask.

**Chemical Properties**—Relatively inert. May contain acetic aldehyde and ether peroxide as impurities due to decomposition which is favoured by air light and heat and retarded by copper diphenylamine and hydroquinone. Thus ether should be stored in dark cool places. The greater the peroxide content, the less potent is the ether as an anæsthetic. Peroxides shown to be present if a yellow colour develops on addition of potassium iodide. Aldehydes in ether will cause Nessler's solution to turn turbid or yellow.

It is unaltered in the body 85 per cent to 90 per cent being eliminated by the lungs. The rate of elimination depends on the tidal exchange and blood flow and can be expedited by inhalation of carbon dioxide.

**Pharmacology—**

**CIRCULATORY SYSTEM**—Heart rate is increased at first due to (1) Adrenaline liberated (2) Sympathetic stimulation and (3) Vagal depression. Later the heart rate is relatively unchanged. A light plane of anæsthesia causes vasoconstriction and a deep one vasodilatation both effects on the vasomotor centre. Return of consciousness causes an increase of tone to a level greater than the original tone—hence the pallor.

Blood pressure not much altered in Plane 2 or above but immediately following induction of anæsthesia the blood pressure is raised due to sympathetic stimulation. Sympathetic block to T<sub>4</sub> abolishes this. If level of anæsthesia is below Plane 2 a fall takes place after the first half hour and is progressive due to depression of smooth muscle in vessel walls depression of skeletal muscle and consequent lack of support to circulation reduced cardiac output and depression of the vasomotor

excreted in the urine. Hyperglycaemia is common and blood sugar levels may be increased two- or three fold. One hour of surgical ether anaesthesia lowers the liver glycogen 50 per cent. It is due to stimulation arising from the midbrain of sympathetic nerves of suprarenal gland causing mobilization of glycogen through extra secretion of adrenaline. If nerves to suprarenal are paralysed no hyperglycaemia is produced by ether. Barbiturate (pentobarbitone) premedication inhibits this hyperglycaemia. morphine does not.

After long periods of ether anaesthesia there is no microscopic change in the liver.

**OTHER EFFECTS**—Body temperature is reduced owing to depression of heat regulating centre and decreased metabolism. The intra-ocular tension is increased.

The spleen is decreased in size up to 50 per cent—another effect of sympathetic stimulation. This action is opposed by barbiturates.

A tremor chiefly affecting the legs is sometimes seen in light ether anaesthesia. Passive stretching of the quadriceps extensors together with deeper anaesthesia will usually abolish these movements.

#### Advantages of Ether —

- 1 It is relatively non toxic and would appear to upset the patient clinically less than biochemical investigation would suggest.
- 2 It will produce excellent relaxation without undue respiratory depression.
- 3 Respiratory depression is not accompanied by serious cardiac damage—in the absence of hypoxia. Artificial respiration will usually overcome the effects of temporary overdosage.
- 4 Thus ether is a very safe anaesthetic. For the unskilled anaesthetist dealing with the unfit patient it has a lot to commend it.

**Light Ether Analgesia**—The advantages of light ether narcosis have been pointed out by Artusio \* who separated the first stage into three planes in the deepest of which he claims to produce true analgesia with a fully conscious and co-operative patient. The technique is recommended by its originator for cardiac surgery.

#### Disadvantages of Ether —

- 1 Its tendency to cause mucus secretion from the upper airway.
- 2 Its tendency to upset the body chemistry.
- 3 Its tendency to irritate the kidneys.
- 4 Its tendency to explode when in contact with sparks flames and hot surfaces.

The concentration in the blood of ether ranges from 100 to 170 mg per cent from light anaesthesia to respiratory failure.

The acoustic gas analyser (Ridley and others *Anesthesiology* 1951 12: 276) shows that the respiratory ether concentration necessary for laparotomy ranges from 6.1 to 13.7 vol per cent.

Light anaesthesia can be maintained by 3 to 6 per cent (25-50 mm Hg partial pressure) in inspired mixture.

\* Artusio J F *J Pharm* 1954 311 343.



Ether—Pharmacology *continued*

neurosurgery Pressure of cerebrospinal fluid consequently rises Electro encephalographic levels of anæsthesia have been described\* and the patterns can be used as a guide to anæsthetic depth Levels 1 and 2 correspond to induction 3 4 and 5 to safe surgical anæsthesia 6 and 7 to unsafe deep levels of anæsthesia There is a constant relationship between E E G patterns and the depth of gas oxygen and ether anæsthesia

Evidence concerning the curare like action of ether on the myo neural junction is contradictory Tendon jerks are nearly always lost during ether anæsthesia seldom in those patients given thiopentone gas and oxygen

**THE SYMPATHETIC NERVOUS SYSTEM**—Central stimulation resulting in (1) Increase of heart rate (2) Increased production of glycogen and a raised blood sugar level (3) Contraction of spleen (4) Dilatation of the gut and inhibition of its movements (5) Bronchial dilatation (6) Dilates coronary arteries

**THE PARASYMPATHETIC SYSTEM**—Central depression

**ALIMENTARY SYSTEM**—Nausea and vomiting occur in more than 50 per cent of patients after ether anæsthesia Salivary glands are stimulated during induction and depressed later

Gastro intestinal atony is produced in deep anæsthesia and post operatively is more marked in the small than in the large bowel This is due to stimulation of sympathetic nerves and atony of plain muscle Liver function decreased but restored to normal within twenty four hours Ether depresses the secretion of bile and bile salts

**THE URINARY SYSTEM**—Urinary flow is diminished because of reduction in plasma volume and renal vascular constriction a neurogenic effect which soon passes off when anæsthesia is stopped

In normal kidneys there is slight reduction of renal function and this is greatly increased in cases of nephritis and is not mediated through the posterior part of the pituitary Both ether and cyclopropane cause renal vascular constriction

**THE PREGNANT UTERUS**—Movements inhibited so that in deep anæsthesia relaxation is good Ether passes the placental barrier and soon reaches the same concentration in foetal blood as in maternal The oxygen carrying capacity of the foetal blood is not altered Ether chloroform and trichlorethylene reduce spasm of the Fallopian tubes

**METABOLISM**—Reduction in plasma bicarbonate and pH of blood and increase in blood carbon dioxide in deep planes The explanation is probably that in deep ether anæsthesia there is overall depression of the respiratory centre which allows increase in alveolar and hence blood carbon dioxide and this further depresses the respiratory centre The importance of adequate ventilation follows manually assisted if necessary Alterations in acid base equilibrium are usually due to alterations in ventilation Ketone bodies may appear and may be

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Ether continued

**Ether Convulsions**—These have been reported with increasing frequency since 1926. They are not due exclusively to ether and may occur during any inhalation anæsthetic. Cause not understood many factors having been held responsible. Examples—Overdosage of ether. Impurities in ether. Excessive temperature of the patient due either to his own illness or to the warmth and humidity of the operating theatre. Carbon dioxide excess. Carbon dioxide lack. Sepsis. Calcium deficiency. Cerebral congestion. Atropine excess. A specific streptococcus producing a neurotoxin situated in the nasopharynx. Many of the convulsions occur in children and young adults.

It has recently been shown by *electro encephalography* that patients who get convulsions are of the convulsion prone type. Some non specific factor associated with the anæsthesia or operation acts as a trigger setting off the convulsions. The triad of depth of ether anæsthesia, hyperthermia and hypercapnia would seem to be the chief causes operating on a convulsion prone nervous system. Experimentally deep ether anæsthesia together with hyperthermia has been used to produce convulsions.\*

Convulsions during anæsthesia may commence as occasional twitching of the muscles of the head and neck or limbs. These may give place to general epileptiform clonic spasms which soon result in deficient tidal exchange from inadequacy of the respiratory muscles. Death may occur from hypoxia. Permanent cerebral damage has been reported in cases recovering from convulsions.

#### TREATMENT —

- 1 See that there is adequate tidal exchange using oxygen. Use artificial respiration through an endotracheal tube if necessary. Withdraw ether.
- 2 Inject intravenously 0.1–0.5 g thiopentone or other barbiturate. Dosage should be just sufficient to control convulsions. Overdosage may produce dangerous depression of the respiratory centre.
- 3 Raise head of table and compress carotids temporarily to reduce blood supply to brain.
- 4 If hyperthermia is in evidence apply cold compresses to patient's limbs and chest and use a fan.
- 5 In addition Kemp recommends intravenous saline with 10 per cent glucose to stimulate renal activity and hence alkali elimination together with instillation via a stomach tube of 2 drachms of ammonium chloride in 2 oz of water.
- 6 Intravenous relaxant e.g. repeated small doses of suxamethonium.

Treatment should be prepared for on the first appearance of muscular twitching. The possibility of convulsions must be borne in mind when ether is being given to patients who are hot. Feverish children should receive little or no atropine.

or scopolamine and are often better anaesthetized with an intravenous barbiturate a relaxant and gas and oxygen. If veins are inaccessible the bone marrow can be used. Thio-pentone can also be given into the rectum.

### DIETHYL ETHER $[(C_2H_5)_2O]$

Known also as divinyl oxide. Proprietary names Vinesthene in Britain Vinethene in the U.S.A. Originally prepared by Semmler in 1887 anaesthetic properties discovered by Leake and Chen in 1930 in an effort to combine the advantages of diethyl ether and ethylene. Used clinically by S. Gelman and Bell of the University of Alberta in 1932.

**Manufacture**—Fusion of  $\beta\beta$ -dichloro-ether with potassium hydroxide using ammonia as a catalyst.

**Physical Properties**—A clear fluid with non irritating odour. Molecular weight 70. Specific gravity 0.77. Boiling point  $28.3^\circ\text{C}$ . Specific gravity of vapour 2.2. Extremely volatile. Oil/water solubility ratio 41:3. Explosive with air between 1.7 per cent and 27 per cent with oxygen between 1.85 per cent and 85 per cent.

**Chemical Properties**—Very unstable. Decomposed by air, light and heat with formation of formaldehyde, acetaldehyde, formic acid and acetic acid. Stored in tightly stoppered coloured bottles containing 4 per cent ethyl alcohol with a trace of phenyl alpha naphthylamine to inhibit oxidation. Put up in bottles of 25 c.c. and ampoules of 5 c.c. and 3 c.c. Unaffected by soda lime.

### Pharmacology—

Elimination unchanged mostly through the lungs rapidly. Light anaesthesia produced by 4 per cent vapour. respiratory arrest if 10 per cent to 12 per cent is inhaled for any length of time after induction. Blood level of 28 mg per cent corresponds with light anaesthesia and 70 mg per cent produces respiratory arrest.

Rapid induction with rapid recovery. Respiratory irritation not so marked as with ether. Nausea and vomiting after operation rare. Anaesthetic potency four times that of ether. Eyeball movements may be active in presence of good muscular relaxation. No cholinergic action. bronchodilatation the only adrenergic effect.

**CIRCULATORY SYSTEM**—Slight cardiac dilatation is seen less than that caused by ether but more than that due to cyclopropane. The following changes have been seen: (1) Auriculo-ventricular nodal rhythm (2) Auricular flutter (3) Ventricular arrhythmia (4) Alteration in sinus rate. Supraventricular displacement of pacemaker has been reported as occurring in 60 per cent of cases. No harmful effect in anaesthetic doses.

**RESPIRATORY SYSTEM**—In light anaesthesia breathing more rapid and shallow than normal. Does not have same stimulant effect on breathing as does diethyl ether nor does it produce the same degree of bronchodilatation. Respiratory centre paralysed before heart.

### Divinyl Ether—Pharmacology *continued*

**ALIMENTARY TRACT**—Motility undisturbed in light inhibited in deep anæsthesia Salivary reaction strongly stimulated in light planes Early relaxation of respiratory muscles

**URINARY TRACT**—Kidneys not irritated they excrete a small amount of the drug Kidney function slightly decreased

**PREGNANT UTERUS**—Labour slowed down in deep not in light anæsthesia Passes the placental barrier During Cesarean section it does not produce a hard tonically contracted uterus and so enables suture to be easily accomplished if a classical operation is performed (Bourne and Williams)

**THE LIVER**—In anæsthesia lasting more than an hour central necrosis of liver lobules has been found Experimentally this is made worse by coexisting hypoxia Vinesthene is less toxic to liver than chloroform but on account of its action on the liver should never be used for longer than forty five minutes The body chemistry is not greatly upset

**NERVOUS SYSTEM**—Convulsions have been reported during and after its use generally when a closed system is employed In children some minutes after the administration of a single dose fits have occurred Abnormal movements are often seen and depend on depth of anæsthesia the greater the depth the more frequent the incidence of abnormal movements Oxygen usually stops them The eye signs are not reliable in assessing the depth of anæsthesia

### Methods of Administration —

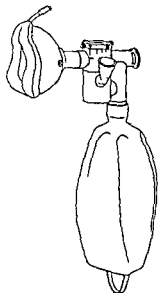
1 **THE OPEN DROP METHOD**—Useful for induction of anæsthesia especially in children A freshly opened bottle should be used and 60–80 drops a minute are sprinkled on to an open mask covered with gauze Owing to its high volatility an even plane of anæsthesia is difficult to maintain Method is wasteful and costly but useful for induction or for short operations Can be given to children for dental extractions by sprinkling it on to gauze placed over the nose (Fig 10)

2 **GOLDMAN'S INHALER**—Specially useful for tonsillectomies with a guillotine and dental extractions in children For children below 5 years old a 3 ml ampoule is used for those above 5 a 5 ml ampoule After shaking the contents of the ampoule into the inhaler the mask is placed on the face and two exhalations are caught in the breathing bag The mask is then firmly applied to the face and to-and fro breathing takes place into the bag Alternatively oxygen or air are fed to the bag through a tap The air breathed vaporizes the vinesthene and in about a minute regular breathing and the loss of the eyelid reflex indicates third stage anæsthesia and the mask is removed Anæsthesia lasts from 50–60 sec If the mask is left too long anæsthesia will lighten as the high concentration of anæsthetic in the brain diffuses into the other body tissues Most patients can leave the theatre under their own power

- 3 **THE OXFORD INHALER** (*Fig 11*)—A modification of Goldman's Inhaler. The vapour concentration can be gradually increased so that the patient does not get the full blast in his first few breaths. Should the bag empty there is an inspiratory valve which allows air to be inhaled and exhaled into the bag.
- 4 **SEMI CLOSED AND CLOSED METHODS**—If vinesthene is put into the chloroform bottle of a Boyle machine it soon vaporizes away. It does so less quickly if put in a Rowbotham's bottle. A drip feed overcomes this difficulty and can be incorporated in the circuit of any standard gas machine. An average of two drops for each inspiration is required. The drug then forms a useful supplement to nitrous oxide and oxygen while it is useful to aid the change-over from nitrous oxide and oxygen to ether.



*Fig 10*—Bellamy Gardner's dropper  
(A Charles King Ltd)



*Fig 11*—Oxford vinesthene inhaler  
(A Charles King Ltd)

- 5 **AS VINESTHENE ANÆSTHETIC MIXTURE**—This was advocated and introduced by Wesley Bourne and consists of 25 per cent vinesthene with 75 per cent ether. The two liquids vaporize at a similar rate and the mixture combines the quick easy induction of vinesthene with the good relaxation and absence of liver damage of ether. Useful for inducing anaesthesia in children either on an open mask or using a machine.

**Advantages of Vinesthene —**

- 1 Quick induction and recovery
- 2 Infrequency of post-operative nausea and vomiting
- 3 Absence of pulmonary irritation
- 4 Requires no heavy apparatus

*Divinyl Ether continued***Disadvantages —**

- 1 Cannot safely be used for long operations
- 2 Causes excessive salivation
- 3 It is explosive
- 4 Owing to its volatility it is wasteful and hard to hold

**Special Indications —**

- 1 For induction of anæsthesia
- 2 For dental extraction and guillotine tonsillectomy in children
- 3 For supplementing nitrous oxide and oxygen anæsthesia in resistant patients
- 4 In obstetrics

**Contra indications —**

- 1 Liver disease
- 2 In presence of sparks or flames
- 3 For long operations

**ETHYL VINYL ETHER [E V E]**

This is a volatile liquid anæsthetic chemically intermediate between diethyl and divinyl ether and has the formula  $C_2H_5-O-C_2H_3$ . It was used by Krantz on animal in 1947\* and is now sold commercially as vinamar. Its vapour density is 2.5 and its boiling point is 35.8°C. It is manufactured by the action of acetylene on ethyl alcohol under pressure in the presence of the catalyst potassium ethylate. Clinically it is more like divinyl than diethyl ether and should be used more for light than for deep anæsthesia† when recovery will be rapid and free from serious sequelæ. It has caused mild convulsions.

**ETHYL CHLORIDE [C<sub>2</sub>H<sub>5</sub>Cl]**

Prepared by Valentine in the seventeenth century and again by Fluorens in 1847 who described its anæsthetic properties which led to investigation by Ernst von Bibra and Emil Harless. First used clinically by Heyfelder of Erlangen Germany in 1848 but was used thereafter for many years only as a local analgesic. Carlson used it as a local spray to ease the pain of dental extraction and succeeded unexpectedly in producing general anæsthesia as the vapour was inhaled. Popularized by McCardie in 1901.

**Manufacture** — By action of hydrochloric acid on ethyl alcohol or ethylene



**Physical Properties** — Clear fluid with ethereal odour. Boiling point is below room temperature 12.5°C. Molecular weight 64. Specific gravity of vapour 2.2. Five volumes of vapour dissolve in one volume of blood. Between 4 per cent and 15 per cent in air will burn with formation of irritating hydrochloric acid fumes. Oil/water solubility ratio high.

Krantz J C, Carr C J, Musser R D and Sauerwald M J *J Pharmacol* 1947 90 83  
 † Dornette, W H L and Orth, O S *Curr Res Anesth* 1955 34 26

Supplied in a pure form for general anaesthesia and in a less pure form with a smaller nozzle for local analgesia. Eau-de Cologne is added to some brands to disguise the odour. The pressure of ethyl chloride in its glass container is 30-40 mm Hg above atmospheric pressure. It can be used as a gas and fed through the rotameter for cyclopropane on the anaesthetic machine when it has little smell and only slight toxic action on the heart. It may like trichlorethylene cause tachypnoea\*.

Cannot be used in closed circuits as it is hydrolysed by the soda lime. Eliminated unchanged from lungs mostly in five minutes.

**Pharmacology**—Potency is 100 per cent and action on the central nervous system similar to that of the other volatile anaesthetics.

The safety margin is small owing to its strength and volatility.

**CARDIOVASCULAR SYSTEM**—Heart rate first decreased a vagus effect later increased. The initial vagal stimulation necessitates that atropine should always be used as premedication. The heart muscle is directly depressed and ventricular fibrillation has been reported; this is extremely rare though Adrenaline may increase myocardial irritability. No electrocardiographic changes were found in 106 patients who received an ethyl chloride induction to ether anaesthesia†.

The blood pressure is decreased due to depression of vasomotor centre. A blood concentration of 20-40 mg per cent corresponds with light anaesthesia to respiratory arrest.

**RESPIRATORY SYSTEM**—The respiratory centre is first stimulated later depressed. It fails before the heart is seriously damaged. Only slightly irritating to mucous membranes but laryngeal spasm not uncommon if drug is administered clumsily. Laryngeal spasm does not occur however during attempts at blind intubation.

**MUSCULAR SYSTEM**—Masseters are liable to go into spasm, an effect which can be overcome by use of the nasopharyngeal airway. Other muscles may show twitching. Convulsions are often seen if the administration is prolonged.

**ALIMENTARY SYSTEM**—Nausea and vomiting are frequent after anaesthesia.

**Signs of Anaesthesia**—Induction may be stormy but gives way in a matter of seconds to the regular breathing and muscular relaxation of surgical anaesthesia. Breathing is deeper than normal and becomes stertorous before overdosage causes it to become progressively hollower. If mask is removed after a few stertorous respirations when amplitude of breathing is maximal about one minute of anaesthesia will result followed by a shorter period of analgesia. or blind nasal intubation can usually be performed especially in children. should the attempt be unsuccessful more ethyl chloride can be given and the manoeuvre repeated. Occasionally as after heavy premedication this respiratory stimulation is not seen. After removal of the mask the patient will become

Cole W. H. J., *Anaesthesia* 1956 11 136.

† Virtue R. W. and Pierce A. F. *Anesthesiology* 1957 12 112.



**Ethyl Chloride—Signs of Anæsthesia** *continued*

a little deeper owing to diffusion into the blood of the vapour still in the air passages and lungs and respiratory arrest has been known to occur even 60–90 seconds after removal of mask. A clear airway is most important and every breath should be heard or seen by the anæsthetist. Apnoea from overdosage must be overcome by artificial respiration. Peripheral vasodilatation usually produces flushing of the face. If this is replaced by pallor anæsthesia may need to be lightened. It is a sign demanding extra caution. If in doubt as to depth of anæsthesia regard the patient as deep until the contrary is proved.

**Methods of Administration**—A prop or mouth gag should always be placed between teeth to facilitate the management of masseter spasm should it arise. The patient should inhale ethyl chloride through the mouth and not through the nose. In this way its unpleasant smell is not noticed nor are its mildly irritating effects on the nose.

- 1 **OPEN DROP METHOD**—The drug is sprayed on to a gauze covered mask gradually but as quickly as it is tolerated. With children the mask can be held some distance from the face by the child itself. As vapour concentration increases the mask is progressively moved nearer the face until unconsciousness is produced. The amounts used vary between 3 ml and 20 ml and vapour strength under mask should be 3.5 per cent to 5 per cent.
- 2 **CLOSED METHOD**—The Goldman or Oxford vinesthene inhalers can be used. The amount of ethyl chloride sprayed into the apparatus varies between 2 ml and 6 ml and depends on the type of patient and length of anæsthesia required. If mask is kept on face after period of maximum respiratory amplitude is reached the level of anæsthesia may become either too deep or too light. The open or closed methods are often used to induce anæsthesia which is to be maintained with ether, the art being to carry the patient from deep ethyl chloride anæsthesia to ether anæsthesia without an intervening period of struggling and interruption of breathing. When the open method is employed one mask should be used for ethyl chloride and a fresh one if ether is to follow.
- 3 **AS SUPPLEMENT TO GAS-OXYGEN ANÆSTHESIA**—A few ml can be introduced into the corrugated tubing of a continuous flow or intermittent flow gas machine to augment the effects of nitrous oxide and oxygen. One plan is to place the ethyl chloride bottle in the breathing bag manipulating its trigger through the rubber from outside.

In resistant or nervous patients having dental extractions under nasal gas-oxygen anæsthesia mouth breathing can be used to get ethyl chloride into the lungs by spraying it on to a towel held over the mouth or on to the mouth pad inside the mouth. With deepening anæsthesia nasal inhalation of the gas and oxygen proceeds but cyanosis must be

- 4 **FOR ANALGESIA**—Ethyl chloride has some use in the production of general analgesia in dental surgery. A special apparatus has been designed for its use.

As a local analgesic ethyl chloride leaves a lot to be desired but is useful before the extraction of loose deciduous teeth in children if sprayed on to the gum.

✓ **Treatment of Collapse**—If this occurs in a young child suspend child by its feet and withdraw tongue while an assistant manually compresses the chest. Sharp slapping of the precordium with a moist towel may stimulate the heart and respiration. If this does not rapidly improve the picture the child should have an airway inserted (if necessary) and the anaesthetist should inflate the lungs either from the reservoir bag of a gas machine or from his own lungs via a face mask. True cardiac arrest must be treated by transthoracic cardiac compression.

#### **Advantages of Ethyl Chloride —**

- 1 Portability
- 2 Rapid induction and recovery
- 3 Economy
- 4 Will not explode though it burns

#### **Disadvantages —**

- 1 Post-operative nausea and vomiting
- 2 Extreme potency and hence potential danger

#### **Indications —**

- 1 For induction before the use of ether. Not frequently employed to-day
- 2 For short operations
- 3 To supplement gas-oxygen anaesthesia in sturdy adults or nervous children e.g. in the dental chair
- 4 In domiciliary work the minimal intravenous thiopentone → open ethyl chloride → blind nasal intubation → open ether sequence can be very useful
- 5 To facilitate blind nasotracheal intubation in children. Absence of laryngeal spasm, deep breathing and speed make this a most useful method.

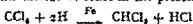
### **CHLOROFORM [CHCl<sub>3</sub>]**

Prepared by Liebig of Germany in 1832 and by Samuel Guthrie in the US and by Soubeiran in France in 1831. Alexandre Dumas described its physical and chemical properties and gave it its name in 1835 because of its relationship to chlorine and formic acid. Anaesthetic properties discovered by Flourens in 1847 and used first in clinical medicine by Simpson and his assistants Mathews Duncan and George Keith in 1847. Within a few months of its discovery it temporarily put ether almost completely out of business. In 1911 Goodman Levy showed that death might be due to ventricular fibrillation in light anaesthesia and not always due to a too concentrated vapour or to overdosage.

A recent book *Chloroform: A Study after One Hundred Years* ed Ralph Waters (Univ of Wisconsin Press Madison 1951) sheds fresh light on the drug.

*Chloroform continued*

**Manufacture**—By action of acetone or ethyl alcohol on bleaching powder. Commercially can be prepared by allowing hydrogen and carbon tetrachloride to react in the presence of iron



**Physical Properties**.—Clear sweet-smelling heavy liquid. Specific gravity of vapour 4.1 (Air = 1). Non flammable but when heated with air in the presence of a caustic or open flame phosgene ( $\text{COCl}_2$ ) is formed. The liquid—though not the vapour—is irritating to skin and mucous membranes. Oil/water solubility ratio is 100. Boiling point  $61^\circ\text{C}$ .

**Chemical Properties**—Although it can be decomposed by alkalis chloroform can be used with soda lime. It is prepared with 1 per cent ethyl alcohol to convert any phosgene formed to diethyl carbonate.

**Pharmacology**—Prolonged inhalation of 2 per cent vapour may produce respiratory arrest but induction will require about 4 per cent. 25–40 mg per 100 c.c. blood = light anaesthesia. 40–70 mg per c.c. blood = respiratory arrest. 20–30 mg per 100 c.c. blood may cause cardiac failure. Thus the safety margin is very slight. It is not altered in the body and is excreted mainly through the lungs. Complete de-saturation takes many hours. Its use is largely given up because of tissue toxicity and action on heart. Its anaesthetic potency is ten times that of ether.

**CARDIOVASCULAR SYSTEM**—Blood pressure gradually falls and decreases with the depth of anaesthesia. On this account alone its use should not exceed one hour. The fall is due to (a) Depressant effect on vasomotor centre. (b) Depressant effect on muscles of vessel walls. (c) Depressant effect on myocardium involving a reduction of its tone, a relaxation of the cardiac walls and an impairment of its functional efficiency (MacWilliam 1890). This may be enhanced by anoxia from respiratory depression. Diastolic blood pressure does not fall as quickly as systolic so that low pulse pressure reduces oozing of blood.

The effect on cardiac rhythm. Arrhythmia much more common than is the case with ether. Heart block and nodal rhythm may occur. Arrhythmia is worst during induction and light anaesthesia. A light plane of anaesthesia causes vasoconstriction and a deeper one vasodilatation due to action on the vasomotor centre and this is reversible by lightening the plane of anaesthesia. The return of consciousness following anaesthesia is accompanied by increase in vascular tone in excess of tone prior to anaesthesia.

Sudden cardiac standstill occurring during light anaesthesia may be due to either (1) Vagal inhibition (2) Ventricular fibrillation or (3) Direct depression of the myocardium. Of these three the first is the least important.

1. **VAGAL INHIBITION**—A sudden inhalation of a strong chloroform vapour may stimulate the vagus and

the so-called trigemino-cardiac reflex. In animals large doses of atropine prevent this. An increase in the sensitivity of the aorticocarotid baroreceptors may be the explanation of vagal inhibition in the induction stage of anaesthesia.\*

2 VENTRICULAR FIBRILLATION—Over half the deaths due to chloroform occur in the first few minutes of induction and are due to this cause. Waters and his colleagues think that this is rare. Deep anaesthesia protects against it. Causes are—

a Irritation of the upper respiratory passages by too strong a vapour may irritate a reflex arc via the fifth nerve brain stem stellate ganglion and cardiac sympathetic nerves. Ventricular tachycardia and extrasystoles may precede the ventricular fibrillation.

b Adrenaline acts on the chloroform sensitized myocardium to produce arrhythmia which may end as ventricular fibrillation. Adrenaline may be autogenous—as from fear or pain due to incision before anaesthesia is complete or it may be exogenous e.g. in nasal packs or along with local analgesic solutions. Thus adrenaline is contra indicated during chloroform anaesthesia.

c Direct effect of chloroform on heart muscle increases irritability of ventricles and reduces the refractory phase of the myocardium. Depression increases with depth of anaesthesia.

d An increase in the blood carbon dioxide level predisposes to ventricular fibrillation. This may easily occur in Stage II anaesthesia following apnoea and a stormy induction.

Under light chloroform anaesthesia 50 per cent of patients develop multiple focus ventricular tachycardia which may give place to fibrillation. It cannot be detected clinically.

Red and white blood-cells are increased in number.

Chloroform prevents the combination of hemoglobin with oxygen as 65 per cent of the drug carried in the blood stream is carried in the red cells because of their high lipid content and chloroform's affinity for lipoids. Oxygen-carrying power of blood is reduced.

CENTRAL NERVOUS SYSTEM—A complete and potent anaesthetic which stimulates the sympathetic nervous system as does ether.

RESPIRATORY SYSTEM—The vapour does not irritate the bronchopulmonary mucosa while the bronchial musculature is relaxed. The respiratory centre is depressed and by the time that respiratory arrest occurs the circulation is so inadequate that efficient artificial respiration may fail to revive it. It is thus often difficult to know which fails first the heart or the breathing.

ALIMENTARY SYSTEM—Nausea and vomiting may occur after operation in 40 to 50 per cent of cases a similar percentage to that caused by ether. The tone and movement of the bowel

\* Robertson J. D. and Swan A. A. B. *Anaesthesia* 1937 12 182

**Chloroform—Pharmacology—Alimentary System continued**

are inhibited during and for some time after anæsthesia. Salivation is increased during induction. The size of the spleen is decreased.

**URINARY SYSTEM**—Urinary Secretion is decreased due to the declining blood pressure. Renal irritation with damage to the tubules may occur giving rise to post-operative albuminuria. Ketonuria and glycosuria may occur. Waters and his co-workers found that renal function as tested by urine analysis and blood investigations such as non protein nitrogen estimation, urea clearance, phenolsulphophthalein tests, blood sugar and alkali reserve estimations is no more depressed after chloroform than after administration of other anæsthetics provided always that hypoxia is avoided. Similarly they found that liver function was not significantly more depressed than in a comparable control series using other anæsthetics.

**PREGNANT UTERUS**—In light planes contractions are not greatly affected but are completely inhibited in deep anæsthesia. Chloroform easily passes through the placental barrier. It is very useful in the production of obstetrical analgesia.

**METABOLIC EFFECTS**—The serum bicarbonate and the pH of the blood are reduced while acetone bodies may appear in the urine. Blood sugar may be increased 200–300 per cent due to mobilization of glycogen from the liver, inhibition of insulin secretion or direct effect on liver cells. The blood sugar returns to normal in twenty four hours. Non protein nitrogen increased.

**LIVER**—The function is decreased with depletion of glycogen and diminished bile formation. Effects can still be found one week after chloroform anæsthesia. While recent work in America by Waters and his colleagues absolves chloroform from blame in the production of liver dysfunction a paper by Sheehan\* tells rather a different tale. He regards the condition of the liver before anæsthesia as the important factor and concludes—  
 (1) If the patient is in a normal metabolic state no liver effects are seen after a short—30 minute—chloroform anæsthetic.  
 (2) If the patient is in normal nutritional state a long anæsthesia or several short ones can produce definite pathological liver conditions observable histologically but seldom causing clinical symptoms other than slight vomiting and perhaps jaundice.  
 (3) If the patient has had prolonged vomiting as after severe vomiting of pregnancy chloroform will always cause necrosis of the central zones of the liver lobules. Clinically some of these patients will die without regaining consciousness others recover consciousness, vomit, become jaundiced about the third day and die a few days later. Prolonged labour and starvation may with chloroform anæsthesia result in this type of liver damage.

**DELAYED CHLOROFORM POISONING**—First described by Casper in 1850. May occur from the first to the third day after anæsthesia.

The symptoms are increasing nausea and vomiting with jaundice prostration and coma and perhaps death. When recovery takes place there is no resulting permanent liver damage.

The condition is a toxic hepatitis like that produced by phosphorus poisoning. There is necrosis of tissue round the veins in the centre of the lobules. Death may occur on the fourth or fifth post-operative day but if the fifth day is survived recovery is usual.

Protection is afforded by a high carbohydrate high protein and low fat diet prior to administration. As hypoxia makes the condition worse it must be rigorously guarded against and additional oxygen given. Sulphonamides may offer some protection.

Treatment consists in pushing fluids intravenously together with plenty of carbohydrate and protein. If necessary these are given intravenously as glucose and amino acids respectively. Methionine may have a place in the treatment.

After one hour of chloroform anaesthesia prothrombin formation by the liver is inhibited and oozing increases.

**Methods of Administration**—Strength of vapour required for induction 2-4 per cent for maintenance 0.2 to 1.5 per cent—average 0.6 per cent (Waters). The patient should be well stocked with carbohydrates and protein before anaesthesia. Morphine may depress respiration and mask signs of anaesthesia so the tyro with chloroform should use it sparingly. A full dose of atropine may help to prevent vagal stimulation. Chloroform sleep must be distinguished from surgical anaesthesia. It is characterized by automatic respiration but the eyelash reflex is present. Hypoxia must be carefully avoided while administration of a little oxygen makes for safety during anaesthesia. Induction is helped by a trickle of carbon dioxide which keeps breathing regular and of equal volume.

Because of the fall of blood pressure chloroform should not be given to sitting patients. It should be administered regularly and not spasmodically so that breath holding followed by hyperpnoea is not produced with its dangers of relative overdose of vapour. A relatively sudden large increase in the concentration of vapour usually precedes cardiac depression and inflation with oxygen will often retrieve the position. Before operation starts depth of anaesthesia should be sufficient to prevent reflex hyperpnoea from surgical stimuli. If struggling or apnoea occurs remove the anaesthetic temporarily so that the patient does not get a sudden lung full of vapour when breathing is resumed.

As one of the chief dangers of chloroform is syncope during induction a small dose of intravenous barbiturate can be used to produce unconsciousness with chloroform used for maintenance.

1. **OPEN DROP METHOD**—One drop of chloroform weighing 20 mg if vaporized in 500 ml of air forms a 1 per cent concentration of vapour in air. Chloroform burns of the eyes and skin must be avoided. One layer of lint on a Schimmelbusch mask (Fig 12) is satisfactory. It is held two inches from the face and is gradually lowered. In the first half minute one drop

**Chloroform—Disadvantages continued**

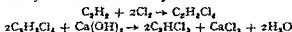
- suddenness This cannot always be avoided even in skilled hands
- 2 It may upset the body chemistry including the liver and should not be used in patients with diabetes ketosis or thyrotoxicosis
  - 3 It is incompatible with adrenaline especially that produced by the patient's emotional response e.g. the frightened healthy young adult
  - 4 A respiratory or circulatory defect may cause delay in desaturation with grave results
- Many experienced anaesthetists now find few real indications for the use of chloroform. If it is used the potentially dangerous induction stage should be accomplished with an intravenous barbiturate while inflation of the lungs with pure oxygen should immediately follow the first sign of circulatory collapse

**TRICHLORETHYLENE [CCl<sub>2</sub>CHCl]**

First described in 1861 by E. Fischer since when it has been used in industry both as a fat solvent and in the dry-cleaning trade. Its poisonous properties have been long recognized (Plessner 1915) especially its power to produce analgesia in distribution of fifth cranial nerve (Oppenheim 1915). On this account it has been used to relieve the pain of trigeminal neuralgia the vapour from 1 ml capsules being inhaled. Relief afforded is probably not a local action but part of a general analgesia. General anaesthetic effects described by Dennis Jackson of Cincinnati in 1933.

Striker used it to anaesthetize 300 patients in 1935 but its present popularity is due to Langton Hewer who published case reports in 1942.

**Manufacture**—Prepared by treating acetylene with chlorine which forms tetrachlorethane and reacts with calcium hydroxide in lime slurry to form trichlorethylene



**Physical Properties**—Colourless liquid with odour resembling chloroform. Specific gravity 1.47. Boiling point high—87°C so volatility is low. Very soluble in lipoids sparingly so in water as oil to water ratio is high. Will not burn or explode under clinical conditions but will ignite in pure oxygen at temperatures above 419°C. May be decomposed into phosgene and hydrochloric acid at temperatures above 125°C. If diathermy is being used in the mouth trlene should be vaporized by air not pure oxygen or nitrous oxide. Vapour nearly five times heavier than air vapour density 4.35 (air = 1).

**Clinical Properties**—Stored in amber glass or aluminium containers as it is decomposed by sunlight. Also decomposed by soda lime if the temperature is above 15°C with formation of toxic products such as dichloroacetylene. Thus the agent must not be used in closed circuits.

For anaesthesia the proprietary form of the drug known as trlene is used. It is coloured blue for identification purposes 1:200,000.

waxoline blue) and contains 0.01 per cent of thymol to retard decomposition. Trilene is stable and does not seriously deteriorate with age. Trimar is another proprietary brand. Trilene may irritate the skin if rubbed on to it e.g. before intravenous injection.

**Pharmacology**—Trichlorethylene resembles chloroform in its effects only it is less depressant to the heart and other organs. It produces good analgesia but usually poor muscular relaxation. It should not be used for deep anaesthesia and if it is pushed rapid breathing or respiratory arrest is likely to occur. Cardiac damage of serious degree seldom precedes respiratory arrest. Concentration in blood of 20–40 mg per 100 ml is required for anaesthesia and for respiratory arrest 100–110 mg per 100 ml.

**CARDIOVASCULAR SYSTEM**—The blood pressure is not greatly altered but oozing from cut surfaces is said to be reduced.

On the cardiac automatic conducting tissue it has well marked effects but these are seldom fatal. Several deaths due to primary cardiac failure have however been reported.\*

Sensitizes heart to effects of adrenaline †

Barnes and Ives† have reported on the electrocardiographic behaviour of the hearts of a group of forty healthy patients under trichlorethylene anaesthesia. They found arrhythmias frequent and divided them into two main groups—

- 1 A group of arrhythmias occurring early in the anaesthesia usually transient and without serious significance probably due to an increase in vagal tone.
- 2 A second group occurring later in anaesthesia taking the form of premature contractions arising from ectopic foci. Pulsus bigeminus was common i.e. a normal beat followed by a premature contraction and a pause. In 10 per cent of their patients a rapid (130–200 a minute) irregular rhythm was noticed indistinguishable clinically from auricular fibrillation but proved to be due to multifocal ventricular tachycardia. Every case of ventricular fibrillation is preceded by this form of arrhythmia so it is potentially dangerous. Adrenaline has the power of converting multifocal ventricular tachycardia into ventricular fibrillation and should not be used together with trilene.

A raised blood carbon-dioxide tension increases the incidence of arrhythmia during trilene anaesthesia while the use of pethidine has the opposite effect ‡. Pethidine also reduces the tachypnoea sometimes associated with trilene anaesthesia as also does reduction of the tension of trilene vapour.

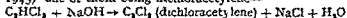
**CENTRAL NERVOUS SYSTEM**—Convulsions have been reported similar to the so called deep ether convulsion. Delay in recovering consciousness after anaesthesia has also been described. There is sometimes a tendency to headache afterwards.

Edwards G, Morton H J V, Pask E A, and Wylie W D. *Anaesthesia* 1956 11 207.  
† Morris L, E, Nottensmeyer M, H, and White J M. *A.esthesiology* 1953 14 153.  
‡ *Poc R Soc Med* 1944, 37 9.  
§ Johnstone M. *Brit med. J* 1951 2 943.



Trichlorethylene—Pharmacology—Central Nervous System *continued*

If trichlorethylene is used in a closed circuit especially with a certain type of soda lime which becomes very hot during use toxic products may be formed (Morton 1943 and McAulay 1943) one of them being dichloroacetylene—



This is a potent nerve poison and may produce paralysis of cranial nerves or even death. The fifth and seventh nerves are most commonly involved but interference with the third fourth sixth tenth and twelfth nerves has been reported. These lesions may be temporary or permanent.

**RESPIRATORY SYSTEM**—As the vapour is not pungent it can be inhaled easily so producing a smooth induction. It does not irritate the respiratory passages or easily produce laryngeal spasm. Increase in concentration of vapour strength can be produced quickly. A small amount of trilene vapour will often settle a patient whose pharyngeal or laryngeal reflexes are active. Thus it probably does by depressing the afferent side of the reflex arc. Tachypnoea which occurs in about one third of patients given trilene results in inefficient tidal exchange and reduced blood-oxygen level. It is due to stimulation of the stretch receptors in the lungs which causes increase in rate and decrease in depth of respiration. Stimulation of deflation receptors produces further increase in respiratory rate\*. Trilene stimulates both these reflexes an effect abolished by vagotomy.

Average rate of breathing with trichlorethylene properly used is between 20 and 30 a minute. If rapid breathing is produced it is a sign of overdosage and the concentration must be reduced until the breathing becomes slow again. Should this not occur a small dose of pethidine (10–20 mg) should be given intravenously or another agent must be substituted. Occasionally sudden respiratory arrest occurs without preliminary tachypnoea this too is a sign of overdosage and can be readily overcome by a little effective artificial pulmonary ventilation. Tachypnoea may occur after relatively little vapour has been given during induction especially in children and for this reason it is often wise to get a patient stabilized with another agent (e.g. thiopentone) using trichlorethylene as a maintenance anaesthetic. The more thiopentone given for induction the less is the likelihood of tachypnoea†. Here a little goes a long way and it finds a very useful field of employment.

**ALIMENTARY SYSTEM**—Post-operative nausea and vomiting are less common than after ether and chloroform. Salivation is not marked.

**MUSCULAR SYSTEM**—Relaxation is not very good. It is useless to push the administration in an effort to obtain this.

**METABOLISM**—The blood chemistry is not greatly altered while the liver is affected less by trilene than it is by ether. In animals very prolonged inhalation of low concentration of

Whitteridge D. and Bulbring E., *Brit med Bull.*, 1946 4 83  
† Dundee J. W. *Brit J Anaesth* 1953 25 1

vapour has failed to cause either anatomical or physiological changes in the organs

**EXCRETION**—A small amount undergoes change in the body with the formation of harmless trichloroacetic acid which is excreted by the kidneys over a period of several days. A diagnosis of trichloroethylene intoxication can be made if more than 7.5 mg of trichloroacetic acid per 100 ml of urine is present. Most of the drug is excreted unchanged by the lungs. A high urinary volume leads to more speedy elimination of trichloroacetic acid while retention of this acid may cause post operative electrolyte imbalance. Infection prolongs the period of elimination and so do large fat deposits. A high basal metabolic rate speeds elimination of the acid. Trichloroethylene addiction leading to psychosis has been reported.

#### Methods of Administration —

- 1 **THE OPEN DROP METHOD**—Low volatility makes this unsuitable but analgesia can be produced in this way and can be followed by open ether e.g. in forceps delivery in obstetrics. 1.5 ml dropped on to a gauze mask will often enable small operations e.g. cystoscopy to be painlessly performed.
- 2 **THE SINGLE DOSE METHOD**—Galley† has described this technique in children using an Oxford inhaler. For dental extractions 1.5 ml are used while for tonsillectomies with the guillotine the dose is 3 ml for children under 5 and 5 ml above that age. The bag should be filled initially with oxygen rather than with the patient's exhalations.
- 3 **THE DRAW OVER METHOD**—Marrett has designed an apparatus whereby the patient's inhalations are drawn over trichloroethylene in a vaporizing bottle. Air enters a one way inlet valve; it is passed through either one or two vaporizing bottles provided with wicks and producing very little respiratory resistance. The amount of vapour is controlled by rotating levers above the bottles. A length of corrugated tubing with expiratory valve and face piece leads from the bottles while there is a tap for either oxygen or ethyl chloride together with a rebreathing bag if partial rebreathing is required. It is usual to put trichloroethylene in one bottle and ether in the other so that the apparatus can be used for many types of operation.
- 4 **THE SEMI CLOSED METHOD**—Trichloroethylene can be placed in the chloroform bottle of a Boyle or McKesson machine but the minimum vaporizing position may deliver too concentrated a vapour. To overcome this the lever is kept off but is occasionally flicked over to on and brought straight back again. Rowbotham's chloroform bottle can be used instead plugged into the circuit (see Fig 71). When the Boyle machine is used combined with the standard reservoir bag and corrugated tube (the Magill rebreathing attachment) and provided that the gases are not bubbled through the trichloroethylene the concentration of vapour inhaled is not likely to exceed 1.5 per cent.

Clifton E. and Goldsmith M. W. *A thesis* 1956 11 28  
 † Call A. H. *Lancet* 1945 2 10

Trichlorethylene—Administration—Semi closed Method *continued*

and cannot be maintained much above 1 per cent \* Vapour strength of 0.5 to 2 per cent is required for light anæsthesia. Administration of trilene vapour should cease well before the end of the operation. The drug forms a useful supplement to nitrous oxide-oxygen anæsthesia a very little making a patient quiet without the need for any hypoxia. This method is *specially helpful for dental extractions given through the nasal inhaler*. Patients thus treated can usually leave the chair as nimbly as after nitrous oxide and oxygen alone.

The Finnie Trilene Vaporizer (*see Fig 32*) is useful for trilene administration for short operations e.g. dental extractions. It is made of metal and employs two ampoules of 6 ml capacity of trilene. A gas mixture can be made to pass directly through the apparatus or through the vaporizing chamber or any intermediate position by the turn of a lever.

Trilene with gas oxygen and intermittent pethidine given intravenously and with atropine as the premedication is a good anæsthetic for operation not involving profound muscular relaxation. Anæsthesia is less precariously light than when gas oxygen and pethidine are used alone.

- 5 IN OBSTETRICS—Trichlorethylene produces excellent analgesia in labour 0.5 per cent w/v in air being a suitable concentration for intermittent inhalation lasting several hours. In some patients drowsiness comes on after three hours requiring reduction of vapour strength to 0.35 per cent. Trilene readily passes the placental barrier. Several methods of vaporization have been devised among them the addition of a Rowbotham's bottle to a Minnitt gas-air machine and Elam's method of mixing 25 per cent trilene with 75 per cent ether in an Oxford vaporizer. Woodfield Davies's modification of Freedman's inhaler (1943) consists of an unspillable bottle which cannot be overfilled. The patient draws air via a face mask and corrugated tube over trilene in the bottle. An inspiratory and an expiratory valve prevent re breathing. It is designed to deliver about 0.6 per cent trilene vapour in air but this is upset by environmental temperature movement of the bottle etc. so that the apparatus has not been accepted as safe for use by midwives working on their own.

Cyprane Inhaler. Another simple apparatus for self administration of trilene in air (*Fig 13*).

Tri-lite Inhaler. Simple in construction and useful for the production of analgesia in minor surgery and obstetrics.

Trigesor. A thermostatically controlled apparatus designed for auto administration of trilene and air.

The Tecota Mark 6 Trilene Inhaler (*see Fig 71*)—Also a temperature-compensated inhaler which weighs in its case about 7 lb. It delivers either 0.5 per cent or 0.35 per cent trilene vapour in air over a range of room temperature between 55 and 95 F. Movement does not alter the vapour concentration.

Epstein Macintosh Oxford Inhaler\* (Emotril) Also for use as an auto analgesia machine in obstetrics. It incorporates a compensating device which for any rise in temperature reduces the amount of vapour leaving the vaporizing chamber. When patient inspires a non return valve opens and air enters through a variable opening into the trilete chamber and emerges mixed with vapour. Another current of room air

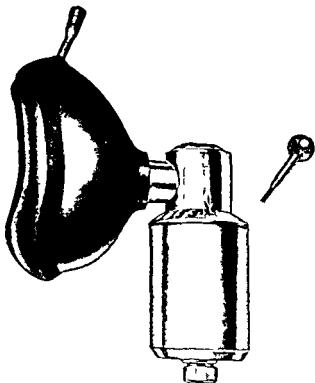
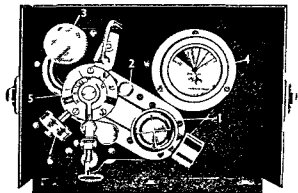
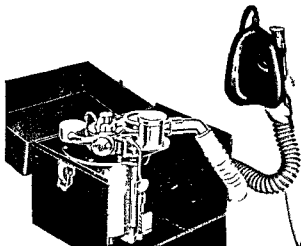


Fig 13—The Cypriote Inhaler (British Oxygen Gases Ltd.)

is drawn through a permanently open by pass of fixed size and dilutes the mixture issuing from the vaporizing chamber. The size of the variable opening can be altered in accordance with the position of a pointer of a thermometer. It is also made with automatic temperature compensator for use by midwives. Normally vapour strength is 0.5 per cent but a small disk can be rotated to make the apparatus deliver a percentage of 0.35. There is a low resistance to respiration and leaks due to an ill fitting face mask are of less consequence.

*Trichlorethylene Administration in Obstetrics continued*

in diluting the mixture reaching the patient than in gas air machines owing to the greater efficiency of trilene vapour as an analgesic (*Fig 14*)



*Fig 14* —The Emotril apparatus (Medical and Industrial Equipment Ltd)

Machines to be used by midwives in Great Britain must fulfil strict conditions. They must deliver trichlorethylene vapour in air at 0.5 per cent and 0.35 per cent concentration with an error of  $\pm 20$  per cent only which must remain constant under all clinical conditions. Each machine must be individually certified and must be periodically checked. Examples are the Tecota Mark 6 and the Emotril Automatic. It is the e

conditions trichlorethylene vapour is as safe as  $\text{H}_2$  and oxygen and more effective. Given with pethidine labour may be prolonged while depression of the infant's respiration may be seen.

#### Advantages of Trichlorethylene —

- 1 Smooth quick induction
- 2 Lack of irritation to upper respiratory tract
- 3 Non flammable
- 4 Portability and economy
- 5 Useful in production of analgesia and light planes of anaesthesia along with nitrous oxide and oxygen e.g. in the dental chair and casualty department
- 6 It is a relatively safe agent for the production of analgesia and light anaesthesia and when so used causes little post operative disturbance

#### Disadvantages —

- 1 Overdosage easy with production of rapid breathing especially in children
- 2 Muscular relaxation inadequate
- 3 Possible effects on heart. Cardiac arrest has been reported but is extremely rare
- 4 Causes more nausea and vomiting than does thiopentone with gas oxygen
- 5 Trilene addiction has been reported and may lead to psychosis
- 6 Cannot be used with soda lime

#### Uses of Trichlorethylene —

- 1 To supplement nitrous oxide-oxygen for light anaesthesia especially when an endotracheal tube or other artificial airway is being used e.g. mastectomy thyroidectomy neurosurgery tonsillectomy minor surgery dentistry etc

To maintain light anaesthesia after it has been instituted with some other agent e.g. thiopentone

- 3 For operations requiring diathermy etc
- 4 As a single dose anaesthetic for short procedures in children
- 5 To produce analgesia in obstetrics

See also Trichlorethylene Anaesthesia by Gordon Ostler, E. S. Livingstone, Edinburgh and London 1953

#### ETHYLENE [ $\text{C}_2\text{H}_4$ ]

This is rarely used in the United Kingdom to day because its advantages over nitrous oxide are very slight while its explosibility is great. Discovered by Johannes Ingenhousz in 1779 or by Becher in the seventeenth century. May have been given as early as 1849 by Nunnally of Leeds.

Anaesthetic properties first noticed by Hermann in 1864. Crocker and Knight (1908) the botanists proved that ethylene contained in illuminating gas would prevent carnation buds from opening. This work was taken up by A. B. Luckhardt and J. B. Carter of Chicago in 1923 and by W. Lasson Brown of Toronto in 1933 and they introduced it into clinical medicine along with Isabella Herb who independently employed it in the Presbyterian Hospital Chicago in March 1933.

**Ethylene continued****Manufacture —**

- 1 Dehydration of ethyl alcohol by sulphuric or phosphoric acid
- 2 Breaking down of propane by heat
- 3 Passing ethyl alcohol and superheated steam over a catalyst such as aluminium oxide

**Physical Properties** — A colourless non irritating gas of unpleasant odour Specific gravity 0.07 so it is lighter than air Boiling point — 103° C. Liquifies at 10° C. under pressure of 60 atmospheres Oil/water solubility ratio 1:4.4 Flammable and explosive with certain proportions of air oxygen and nitrous oxide Explosive range 2 per cent to 28 per cent with air 2 per cent to 80 per cent with oxygen

Eliminated from the lungs unchanged the greater part in two minutes Not altered by soda lime

Analgesia requires from 0 per cent to 35 per cent Anæsthesia from 80 per cent to 90 per cent

**Pharmacology** — Ethylene has almost no effect on the body's metabolism other than causing a slight rise in blood sugar and an inhibition of bile acid secretion

**Induction** — More rapid than with nitrous oxide no stimulation of respiration Mucous and salivary secretions not increased

**Maintenance** — Ethylene is more powerful than nitrous oxide because of its higher oil/water solubility ratio It is less powerful than cyclopropane and ether Anæsthesia differs from that due to nitrous oxide in that —

- a Muscular relaxation is greater
- b More oxygen can be used so that cyanosis does not play such a part
- c Breathing is quieter

Anæsthesia can be carried to lower Plane 1 About 10 per cent of oxygen can be given during induction and when patient is settled and well premedicated up to 20 per cent oxygen will not lighten the anæsthesia

Recovery is rapid but post operative nausea and vomiting are more frequent than after nitrous oxide

It does not depress the fetal respiratory centre or interfere with uterine contractions

**Methods of Administration** — Can be used in the same way as nitrous oxide If the smell is objected to induction can be carried out with nitrous oxide with maintenance by ethylene otherwise a few inspirations of ethylene are taken and then 5 per cent oxygen added and a larger percentage gradually worked up to The closed circuit technique with carbon dioxide absorption lessens the chances of explosion As with nitrous oxide with this technique nitrogen must be removed from the bag from time to time Ethylene can be supplemented with ether cyclopropane or thiopentone

**Advantages of Ethylene**—Ethylene offers the same advantages as nitrous oxide—non toxicity rapid induction and recovery with the additional ones of producing greater muscular relaxation with less risk of hypoxia

**Disadvantages**—Its explosiveness It slightly increases capillary oozing when compared with nitrous oxide

### NEOTHYL

(Metopryl)

This is methyl  $\Delta$  propyl ether  $\text{CH}_3\text{CH}_2\text{CH}_2\text{CH}_3 \diagup \text{O}$

First described by Chancel in 1869

First used by Krantz (1946) Neothyl is coloured green for identification Can be used in closed circuits It contains 0.002 per cent of diphenylamine to prevent decomposition Sold in 150 and 600 ml containers

It has been used in anaesthesia and is similar to diethyl ether but differs from that agent in being less irritant to the respiratory tract causes less nausea and vomiting and is more potent and has a wider margin of safety Boiling point 39 C Not metabolized in body Rapidly eliminated Explosive Has no harmful action on the myocardium although it lowers blood pressure and causes bradycardia Does not sensitize myocardium to effects of adrenaline Has no effect on liver or kidneys but raises blood sugar rather more than does cyclopropane although less than ether A good analgesic not a good muscle relaxant Has an unpleasant smell Has been recommended for use in minor surgery instead of trilene\* to supplement nitrous oxide and oxygen Causes less nausea and vomiting but more salivation than trilene and also produces a greater degree of muscular relaxation Useful for induction of anaesthesia before ether is given as it does not readily stimulate coughing

Does not greatly stimulate rate or depth of respiration It produces a smooth and rapid recovery from anaesthesia which is followed by a prolonged period of analgesia A rapid increase of vapour strength during induction causes hypotension

### ISOPRYL

This is isopropyl methyl ether  $\text{CH}_3\text{CH}(\text{CH}_3)\text{OCH}_3$  and behaves similarly to metopryl

### ISOPROPYL CHLORIDE

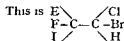
(Propofesin)

This gives a pleasant induction and patients can be carried into deep anaesthesia without struggling It exerts however a toxic effect on the myocardium irregularities of the pulse being common A death has been reported following its use



## HALOTHANE

(Fluothane)



(1 bromo 2 chloro 1,1,1 trifluoroethane)

**History**—Synthesized in the laboratories of Imperial Chemical Industries near Manchester by C. W. Suckling in 1951 and studied pharmacologically by J. Raventos in 1956. Used clinically by M. Johnstone of Manchester followed by Bryce Smith and O'Brien of Oxford. Because of its potency its use calls for great care.

**Physical Properties**—A colourless liquid volatile anaesthetic with specific gravity 1.860 (ether is 0.713) boiling point 50°C vapour pressure at 20°C 743. Has a characteristic odour. Decomposed by light (unless stabilized by 0.01 per cent thymol) but is stable when stored in amber coloured bottles. Is not decomposed by soda lime. In the presence of moisture it attacks tin and aluminium in vaporizers. Non flammable and non explosive when its vapour is mixed with oxygen in concentrations between 0.5 and 50 per cent. Should not be used in circle absorbers because of corrosive action on washers.

**Pharmacology**—As an anaesthetic it is four or five times as potent as diethyl ether and one and a half times as potent as chloroform. Vapour is pleasant to smell and is non irritant. For induction of anaesthesia 2–4 per cent vapour is necessary for maintenance 1–2 per cent. It is a very potent agent and has already caused several deaths from cardiac failure probably because of overdosage.

**CARDIOVASCULAR SYSTEM**—The Medical Research Council Committee on Non-explosive Anaesthetic Agents reports that halothane depresses the myocardium and reduces cardiac output depresses the central vasomotor mechanism and has a small peripheral ganglionic blocking effect. Other investigators stress the last of these effects rather than the first two. There is minimal ECG evidence of increased irritability of the myocardium when low concentrations are used with higher concentrations (which are clinically unnecessary) a vagal type of arrhythmia is sometimes seen. Most arrhythmias are ventricular extrasystoles. Blood pressure falls in proportion to the concentration of vapour inhaled but is always present in some degree. The heart is sensitized to adrenaline although small volumes (0.1 ml) of 1:150,000 solution have been injected subcutaneously without harm. Intravenous noradrenaline may result in ventricular fibrillation. Bradycardia usually results but can be controlled by atropine. There is vasodilatation of both peripheral and splanchnic vessels but during surgery bleeding is reduced and postural hypotension is readily produced. The ischaemia may be more dangerous than that caused by drugs having their sole effect on the autonomic ganglia.

**ALIMENTARY SYSTEM**—The secretion of saliva, mucus and gastric juice is not stimulated while post operative nausea and vomiting are seldom severe.

**CENTRAL NERVOUS SYSTEM**—It is a total anesthetic like ether or chloroform but not a good analgesic.

**AUTONOMIC NERVOUS SYSTEM**—It depresses the sympathetic nervous system after the manner of a high extradural or spinal analgesia. It depresses conductivity through the peripheral sympathetic ganglia and potentiates hexamethonium and d-tubocurarine. Transmission through vagal ganglia is unimpaired. Para sympathetic Stimulant.

**RESPIRATORY SYSTEM**—A respiratory depressant. Respiratory rate often increased and depth decreased effects controllable with pethidine. Induction by fluothane and air may require ventilation and assisted breathing may be necessary. The drug causes bronchodilatation and is very suitable for patients with bronchospasm, emphysema and chronic bronchitis. Not a bronchial irritant. Pharyngeal and laryngeal reflexes depressed early and secretions not stimulated. Because of respiratory depression at least 50 per cent of oxygen should be routinely administered. Care must be taken that controlled breathing does not give rise to inadequate cardiac filling; otherwise severe hypotension may be caused. Control of respiration is easy with halothane.

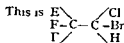
**MUSCULAR SYSTEM**—Moderate relaxation is produced by anæsthetic concentrations and the masseters are relaxed early, making laryngoscopy easy. Full abdominal relaxation is not seen with safe (1-2 per cent) concentrations.

**LIVER AND KIDNEYS**—No serious damage has been reported. A diuresis may be seen after operation.

**INFLUENCE ON SHOCK SYNDROME**—Shock appears to be prevented and hypotension associated with tachycardia, pallor and sweating and vasoconstriction due to overactivity of the sympathetic nervous system is not seen in patients anæsthetized with fluothane. Blood lost must be replaced quickly and sudden movement of the patient may cause trouble as the compensating vasoconstrictor mechanism is absent. Severe hypotension is often greatly improved by the intravenous injection of methoxamine.

**USE WITH MUSCLE RELAXANTS**—The myoneural effect of d-tubocurarine is moderately potentiated while its somewhat weak ganglionic blocking effect is very markedly potentiated so that the two agents should not be used together. The former of these effects is seen with gallamine but as this agent has no ganglionic blocking effects the combination of the two drugs is satisfactory and may, because of the specific effect of gallamine in paralysing the ability of the vagus to slow the heart, make it the relaxant of choice. Halothane somewhat antagonizes the effects of suxamethonium but this is not important clinically.

**Premedication**—Atropine  $\frac{1}{4}$  gr.  $\frac{1}{2}$  gr. or even more. This can be given just before induction intravenously or it may be combined with thiopentone. If necessary pethidine 50 mg. can be given in addition. Heavy doses of respiratory depressants should be avoided.

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**ALIMENTARY SYSTEM**—The secretion of saliva mucus and gastric juice is not stimulated while post operative nausea and vomiting are seldom severe.

anaesthesia and hypotension are required as in neurosurgery throat and nose surgery plastic surgery etc

The newcomer to this drug is advised to avoid too high a concentration to ensure proper ventilation by assisted respiration if necessary to use plenty of atropine to replace blood which is lost to treat undue hypotension with methoxamine and to keep the state of the circulatory system under constant observation

**Vaporizers for Halothane**—Because of its potency accurate and fine control of the vapour strength inhaled is required and for this purpose the Fluotec Vaporizer has been designed (Cyprane Ltd) It remains accurate despite changes in temperature the passage of time the amount of liquid in the container and the gas flow provided that between 4 and 16 litres a minute are supplied to it It is individually calibrated to deliver 0.5 per cent to 3.0 per cent halothane vapour by volume with a gas flow ranging between 4 and 16 litres per minute The calibrations are at  $\frac{1}{2}$  per cent intervals The automatic temperature compensating valve ensures accuracy of vapour concentrations at all temperatures between 13 and 32 C The vaporizer is designed to fit the ordinary Boyle machine but can be adapted to other makes (See also Johnstone M *Brit J Anaesth* 1956 28 392 Raventos J *Brit J Pharmacol* 1956 11 394 Bryce Smith R and O'Brien H D *Brit med J* 1956 2 969 Brennan H J Hunter A R and Johnstone M *Lancet* 1957 2 453 Report of Medical Research Council Committee on Non Explosive Anaesthetics *Brit med J* 1957 2 479)

## FLUOROMAR

This new volatile agent trifluoro ethyl vinyl ether ( $\text{CF}_3-\text{CH}_2-\text{O}-\text{CH}=\text{CH}_2$ ) was described by Lu and Krantz and investigated by Krantz in 1953

**Physical Properties**—It is a stable clear fluid with boiling point of 42.7 C It is not altered by hot soda lime Its oil/water solubility is 94 Its lower limit of flammability in oxygen and in nitrous oxide-oxygen (75:25) is 4 per cent and concentrations greater than 4 per cent will explode

**Pharmacology and Clinical Uses**—Because of its high boiling point it is difficult to induce anaesthesia on the open mask It does not sensitize the heart to adrenaline nor does it produce dangerous arrhythmias Deepening the anaesthesia sometimes causes a fall in blood pressure While the induction period when it is added to gas and oxygen is relatively short the recovery period is relatively rapid Jaw relaxation may be difficult to produce It is less irritating than ether to the air passages but may like cyclopropane cause apnoea at light levels of anaesthesia and like trichlorethylene it may give rise to tachypnoea which can be controlled by small doses of pethidine The anaesthesia it produces cannot easily be fitted into the Guedel pattern and it may be most difficult to assess anaesthetic depth The amount of abdominal relaxation it produces is variable but so far no ill effects have resulted from its combination with relaxants

*Halothane continued*

**Induction**—This may be by the gas-oxygen-halothane sequence or may be by intravenous thiobarbiturate 150–250 mg. The masseters relax early and the patient soon tolerates an airway and shortly afterwards allows an endotracheal tube to be inserted. Salivary and mucus secretion are not stimulated and coughing and laryngeal spasm are rare. Because of its high cost the drug can be given in a Waters circuit with a gas flow of 2–4 litres a minute and a leaking expiratory valve using an accurate vaporizer. A tightly closed circuit should not be used because a dangerously high concentration of vapour may be built up in it. When Boyle's machine is employed a flow rate of 5 litres a minute of gas and oxygen with 50 ml. of halothane in the smaller vaporizing bottle and the plunger at the top gives a range of concentrations from 0.1 to 2.8 per cent. If the plunger is 3 mm. above the surface of the drug the concentration varies between 0.6 and 4.8 per cent. An accurate vaporizer is necessary but if the Boyle machine is used the indicator should seldom go beyond the third mark.

**Maintenance**—Requires 1–2 per cent vapour concentration with at least 50 per cent oxygen. Respiration during a lower laparotomy should usually be spontaneous but may need to be assisted or controlled. A relaxant will usually be required for the initial abdominal exploration as well as for the closure. Succinylcholine well diluted in 50 mg. doses may be used for this but fasciculation is marked even after repeated injections. Upper laparotomies may require a relaxant and controlled breathing. Tachypnoea can be controlled by pethidine and hypotension by 5 mg. doses of methoxamine given intravenously. Arrhythmias do not follow the use of this agent. d-tubocurarine should not be used as it causes hypotension but gallamine in 20–40 mg. doses may be safely used even though its effects and duration are potentiated. Its specific action in paralysing the ability of the vagus to slow the heart may be specially useful during anaesthesia with fluothane. The sensitivity of the heart to the inhibitory effects of neostigmine is increased and if this anticholinesterase must be used it should be carefully preceded by adequate amounts of atropine.

After operation there may be a diuresis and shivering, a dry mouth and diplopia have been reported. Nausea and vomiting are not as a rule troublesome.

Halothane potentiates the action of insulin but does not greatly alter the blood sugar level. It increases the effects of hexamethonium and should not be used with chlorpromazine or other adrenolytic agents. Halothane makes the patient very susceptible to blood loss as it abolishes the compensatory vasoconstrictor mechanisms. Blood should be given as it is lost and methoxamine used if necessary.

**Indications**—This is a very new agent and is not the final solution to the problem of finding a non-flammable volatile anaesthetic as safe as ether. It needs care and vigilance during its administration. It has proved useful in patients with bronchitis, bronchospasm and emphysema. It has found a place when light

anaesthesia and hypotension are required as in neurosurgery, throat and nose surgery, plastic surgery, etc.

The newcomer to this drug is advised to avoid too high a concentration to ensure proper ventilation by assisted respiration if necessary, to use plenty of atropine to replace blood which is lost to treat undue hypotension with methoxamine and to keep the state of the circulatory system under constant observation.

**Vaporizers for Halothane**—Because of its potency, accurate and fine control of the vapour strength inhaled is required, and for this purpose the Fluotec Vaporizer has been designed (Cyprane Ltd). It remains accurate despite changes in temperature, the passage of time, the amount of liquid in the container, and the gas flow provided that between 4 and 16 litres a minute are supplied to it. It is individually calibrated to deliver 0.5 per cent to 3.0 per cent halothane vapour by volume with a gas flow ranging between 4 and 16 litres per minute. The calibrations are at  $\frac{1}{10}$  per cent intervals. The automatic temperature compensating valve ensures accuracy of vapour concentrations at all temperatures between 13 and 32°C. The vaporizer is designed to fit the ordinary Boyle machine but can be adapted to other makes. (See also Johnstone M. *Brit J Anaesth* 1956 **28** 392; Raventos J. *Brit J Pharmacol* 1956 **11** 394; Bryce-Smith R. and O'Brien H. D. *Brit med J* 1956 **2** 969; Brennan H. J., Hunter A. R. and Johnstone M. *Lancet* 1957 **2** 453; Report of Medical Research Council Committee on Non-Explosive Anaesthetics *Brit med J* 1957 **2** 479.)

## FLUOROMAR

This new volatile agent, trifluoro ethyl vinyl ether ( $\text{Cl}_3-\text{CH}_2-\text{O}-\text{CH}=\text{CH}_2$ ) was described by Lu and Krantz and investigated by Krantz in 1953.

**Physical Properties**—It is a stable, clear fluid with boiling point of 42.7°C. It is not altered by hot soda lime. Its oil/water solubility is 94. Its lower limit of flammability in oxygen and in nitrous oxide-oxygen (75:25) is 4 per cent and concentrations greater than 4 per cent will explode.

**Pharmacology and Clinical Uses**—Because of its high boiling point it is difficult to induce anaesthesia on the open mask. It does not sensitize the heart to adrenaline nor does it produce dangerous arrhythmias. Deepening the anaesthesia sometimes causes a fall in blood pressure. While the induction period when it is added to gas and oxygen is relatively short, the recovery period is relatively rapid. Jaw relaxation may be difficult to produce. It is less irritating than ether to the air passages but may, like cyclopropane, cause apnoea at light levels of anaesthesia and like trichlorethylene it may give rise to tachypnoea which can be controlled by small doses of pethidine. The anaesthesia it produces cannot easily be fitted into the Guedel pattern and it may be most difficult to assess anaesthetic depth. The amount of abdominal relaxation it produces is variable but so far no ill effects have resulted from its combination with relaxants.

*Halothane continued*

**Induction**—This may be by the gas-oxygen-halothane sequence or may be by intravenous thiobarbiturate 150–250 mg. The masseters relax early and the patient soon tolerates an airway and shortly afterwards allows an endotracheal tube to be inserted. Salivary and mucus secretion are not stimulated and coughing and laryngeal spasm are rare. Because of its high cost the drug can be given in a Waters circuit with a gas flow of 2–4 litres a minute and a leaking expiratory valve using an accurate vaporizer. A tightly closed circuit should not be used because a dangerously high concentration of vapour may be built up in it. When Boyle's machine is employed a flow rate of 5 litres a minute of gas and oxygen with 50 ml of halothane in the smaller vaporizing bottle and the plunger at the top gives a range of concentrations from 0.1 to 0.8 per cent. If the plunger is 3 mm above the surface of the drug the concentration varies between 0.6 and 4.8 per cent. An accurate vaporizer is necessary but if the Boyle machine is used the indicator should seldom go beyond the third mark.

**Maintenance**—Requires 1–2 per cent vapour concentration with at least 50 per cent oxygen. Respiration during a lower laparotomy should usually be spontaneous but may need to be assisted or controlled. A relaxant will usually be required for the initial abdominal exploration as well as for the closure. Suxamethonium well diluted in 50 mg doses may be used for this but fasciculation is marked even after repeated injections. Upper laparotomies may require a relaxant and controlled breathing. Tachypnoea can be controlled by pethidine and hypotension by 5 mg doses of methoxamine given intravenously. Arrhythmias do not follow the use of this agent. *d*-tubocurarine should not be used as it causes hypotension but gallamine in 20–40 mg doses may be safely used even though its effects and duration are potentiated. Its specific action in paralysing the ability of the vagus to slow the heart may be specially useful during anaesthesia with fluothane. The sensitivity of the heart to the inhibitory effects of neostigmine is increased and if this anticholinesterase must be used it should be carefully preceded by adequate amounts of atropine.

After operation there may be a diuresis and shivering, a dry mouth and diplopia have been reported. Nausea and vomiting are not as a rule troublesome.

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**Indications**—This is a very new agent and is not the final solution to the problem of finding a non-flammable volatile anaesthetic as safe as ether. It needs care and vigilance during its administration. It has proved useful in patients with bronchitis, bronchospasm and emphysema. It has found a place in light

Durasorb is an improved soda lime with a prolonged effective life which does not overheat. Its pink colour turns to white when it becomes inactive.

Baralyme is 80 per cent calcium hydroxide with 20 per cent barium hydroxide. It is said to be less caustic and to produce less heat than soda lime. No silica is necessary to produce hardness.

Amounts of carbon dioxide up to 2 per cent may be present in the respirable atmosphere without being detectable by clinical observation on the patient's tidal volume, respiratory rate, pulse or blood pressure changes and may cause an unsuspected respiratory acidosis. It has however been stated that 0 per cent is the highest permissible concentration of carbon dioxide in an anaesthetic circuit. It is therefore most important to see that the soda lime is fresh and that tidal exchange is adequate for efficient ventilation. There is a tendency for gases to flow so that more absorption takes place in the soda lime in contact with the walls of the canister than in that in the middle.

#### SIGNS OF EXHAUSTION OF SODA LIME —

- 1 Rise in B.P. followed eventually by a fall
- 2 Rise in pulse rate
- 3 Deepening of respiration
- 4 Increased oozing from wound
- 5 Increase in volume of gases in breathing bag, a late sign which should never occur—about 200 ml per minute are excreted. Some brands change colour when exhausted but this is not a reliable sign. The one pound canister will last about six hours intermittently, two hours continuously. In practice it is unwise to wait until the so-called signs of exhaustion of the soda lime appear. Fresh absorbent should always be used if there is any doubt as to its efficiency.

#### Advantages —

- 1 Quiet breathing, controllable in rate and depth
- 2 Economy in use of gases
- 3 Conservation of the patient's heat and water vapour
- 4 Less risk of explosion as no gas escapes into the surrounding atmosphere

#### Disadvantages —

- 1 Tight fit of mask, tube etc. to patient may cause trauma
- 2 Alkaline dust may pass to patient
- 3 Heat from chemically active soda lime may cause sweating
- 4 Resistance to breathing and dead space are high. The continued increase of inspiratory resistance may give rise to an increase of the negative intrapulmonary pressure and acute pulmonary oedema while continued expiratory resistance may cause a positive intrapulmonary pressure increase with increased peripheral venous pressure and decreased cardiac output. These factors while relatively unimportant in the fit may be harmful in the ill, the old or the very young patient.
- 5 Results not always as good as when an open circuit is used.



**Fluoromar—Pharmacology and Clinical Uses** *continued*

Surgical anaesthesia is present when the blood level is between 17 and 38 mg per cent and alveolar gas levels between 3 and 8.2 vols per cent (See also Lu G Ling J S L and Krantz J C jun *Anesthesiology* 1953 **14** 466 Sadoy M S Balagot R C and Linde H W *Ibid* 1956 **17** 591 Dundee J W Linde H W and Dripps R D *Ibid* 1957 **18** 66 Dundee J W *Proc R Soc Med* 1957 **50** 191)

**CHAPTER VII****THE CLOSED CIRCUIT, CYCLOPROPANE, CONTROLLED RESPIRATION****THE CLOSED CIRCUIT WITH CARBON DIOXIDE ABSORPTION**

Introduced by John Snow in 1850 revived by Dennis Jackson in 1915 for work on animals used by Waters of Madison in 1900 in clinical anaesthesia and first reports appeared in 1924 The circle or two phase system was devised by Brian Sword in 1926 W B Frimrose of Glasgow used caustic soda solution as an absorber in 1931 while Dräger patented an apparatus with a closed circuit in 1926

Founded on principle that if sufficient oxygen is added to supply body's basal needs and carbon dioxide is absorbed the same mixture of gases can be used repeatedly as it is exhaled unchanged Basal oxygen varies between 200 and 400 ml per minute so that closed-circuit anaesthesia can only be used with a machine capable of delivering accurately measured small volumes of gases The completely closed circuit is not employed as frequently to day as it once was unless cyclopropane is the anaesthetic agent Instead a high rate of gas flow with an escape of gas during expiration is arranged the soda lime removing any carbon dioxide which remains in the circuit

**Soda lime**—Used to absorb the carbon dioxide A mixture of 90 per cent calcium hydroxide with 5 per cent sodium hydroxide with silicates to prevent powdering It is a 10 per cent aqueous solution of sodium hydroxide dispersed on calcium hydroxide These hydroxides combine with carbon dioxide in the presence of water to form carbonates Wilson soda lime the type used in anaesthesia is specially prepared its granules are size 4-8 mesh to minimize resistance to breathing and to allow plenty of surface for absorption Air space in the charged canister should equal the patient's tidal volume The chemical change involved in absorption results in heat production the heat of neutralization Some regeneration of activity occurs if exhausted soda lime is rested for two hours Storing soda lime in its container does not interfere with its efficiency

canister has been described\* which weighs 8 ounces less than the metal one is just as efficient and gets no hotter. Resistance to gases 2-3 cm water. In the to and fro system the pressure in the face piece varies from +0.75 cm of water to -0.75 cm of water.

- 2 The circle or two-phase system (Brian Sword). An inspiratory and an expiratory tube are used with flap valves to ensure a one way flow of gases. Breathing is thus divided into two phases and dead space is minimized. Efficiency is lost if the tidal volume is greater than the air space between granules. The soda lime can be by passed and the canister can be easily removed for recharging. The gases can be passed through or over ether. The reservoir bag is either the bladder or concertina type. In this system the canister does not drag on the mask and there is less heating of inspired gases. Resistance to gases 2-3 cm water. In the circle absorber face piece the pressure varies from +2 cm of water to -1.75 cm of water.

Circuits on the Mushin Coxeter (1941) Boyle Absorber Mark II (Fig 16) McKesson M I E and Armed machines are of this type. Circle and to and fro absorbers are equally efficient if properly designed although resistance to respiration is nearly twice as great in the circle as in the Waters system because of the corrugated tubing and valves.

**Dead Space**—The space occupied by gases not freed of carbon dioxide is called mechanical dead space. That space occupied by gases—e.g. pharynx, larynx, bronchi, etc.—which do not come into contact with the alveolar epithelium.

Mechanical dead space in Waters absorber is space between the face and the wire gauze of the canister. With a large mask this may be 100 ml.

The use of a simple nylon pot scourer in the Waters canister has been recommended†. This should be inserted after the canister has been filled and shaken down as lightly as possible and its presence reduces the amount of carbon dioxide in the bag during use.

Mechanical dead space in circle absorber is space between face and beginning of double corrugated tubing. A small mask reduces it—and so does an endotracheal tube connected directly to the anaesthetic breathing tubes.

Cope's modification of Waters absorber is designed to minimize dead space when anaesthetizing children (Fig 17). In the Coxeter Mushin machine gases pass through soda lime during both inspiration and expiration.

**CARBON DIOXIDE ACCUMULATION**—Respiration normally keeps the alveolar carbon dioxide concentration at 5.6 per cent which gives a blood carbon-dioxide tension of 40 mm Hg. If the tidal exchange is reduced by one third the alveolar concentration of the gas doubles whereas a tidal exchange of more than 75 per cent above normal is required to halve the carbon

\* Kilpatrick L. G. *Anaesthesia* 1951 6 236  
 † Robson J. G. and Park E. A. *Br J Anaesth* 1954 26 333

## Carbon Dioxide Absorption—Disadvantages continued

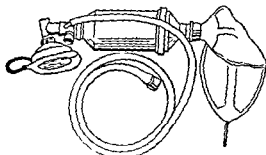
- 6 Accumulation of water vapour It interferes with heat loss and may lead to sweating during operation
- 7 Increased carbon dioxide content of respired gases as absorption is far from perfect
- 8 Dilution of gases in reservoir bag by nitrogen

Cross infection from one patient to another is possible and some machines incorporate a water bacterial trap

The present awareness of carbon dioxide accumulation in anaesthetic circuits has led many observers to question the efficiency of the closed circuit system as a sufficiently reliable method of carbon dioxide absorption \* Monoethanolamine has been suggested as a substitute for soda lime in closed circuits †

## Apparatus —

- 1 The Waters to and fro single phase system (Fig 15) This consists of a mask separated from the breathing bag by a canister of soda lime Gases pass through the canister during both inspiration and expiration Fresh gases are led to the patient



F 15 —The Waters to-and-fro carbon dioxide absorber  
(British Oxygen Co. Ltd.)

close to the mask. Waters canister was designed after much experimenting as to shape and size. It measures 12 cm long by 8 cm in diameter and holds 1 lb of soda lime. The air space between granules averages 400 ml. To obtain maximal efficiency the tidal exchange should approximate the air space of the charged canister. If the tidal volume is greater than the air space the gases may pass through too rapidly for efficient absorption. If it is less than the air space as when a large canister is used on a child or when breathing is depressed absorptive efficiency is sacrificed because the soda lime in the front of the canister becomes exhausted as the gases come into intimate contact with this part only. In such circumstances a smaller canister should be used. It is heavy and rather awkward to use but is cheap and can easily be sterilized. A rubber

dioxide concentration \* No matter how efficient the soda lime carbon dioxide will not be absorbed unless the tidal exchange is normal or above normal During controlled breathing a too energetic artificial ventilation will result in a poorer absorption †

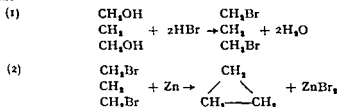
Respiratory acidosis—greater than acidosis produced in any other way—can be caused if the blood carbon-dioxide is allowed to rise to 100 mm Hg

Circulatory collapse may occur after operation when carbon dioxide has been allowed to accumulate from stale soda lime or from hypoventilation and the latter may occur after the patient has left the care of the anaesthetist

### CYCLOPROPANE

Cyclopropane or trimethylene was first synthesized by v. Freund in 1882 its anaesthetic properties were shown by G. H. W. Lucus and V. E. Henderson of Toronto in 1929 clinical reports by Waters and his colleagues followed in the years after 1930 when he started its use (Stiles, Neff, Rovenstine and Waters 1934) Its pharmacology was worked out by Seevers, Meek, Rovenstine and Stiles in 1934

A saturated hydrocarbon isomeric with propylene In Britain prepared from trimethylene glycol in the production of glycol from the fermentation of molasses Trimethylene glycol reacts with hydrobromic acid to form trimethylene dibromide which is then reduced by zinc



Is also prepared from natural gas found in the U.S.

**Physical Properties**—Colourless gas with sweet smell Molecular weight 42.05 Boiling point  $-32.9^\circ\text{C}$  One and a half times heavier than air Liquefies at ordinary temperatures if pressure of 5 atmospheres is applied hence is stored in light alloy cylinders as a liquid no reducing valves being required One ounce is equivalent to 3.5 gallons (4.29 gallons U.S.) Almost insoluble in water (1 volume dissolves in 2.7 volumes at  $15^\circ\text{C}$ ) but very soluble in lipids oil/water ratio 34.4 oil/blood ratio 15.3 Very explosive with oxygen (between 2.5 and 50 per cent) and nitrous oxide throughout the anaesthetic range explosive in air between 3 per cent and 10 per cent Propylene is the chief impurity and is harmless in small volumes The gas is supplied in aluminium alloy cylinders orange in colour

Orto, R. H. *Anaesthesia* 1952 7 4

† Lund, I. Lund, O. and Erikson, H. *Brit J Anaesth* 1957 29 17

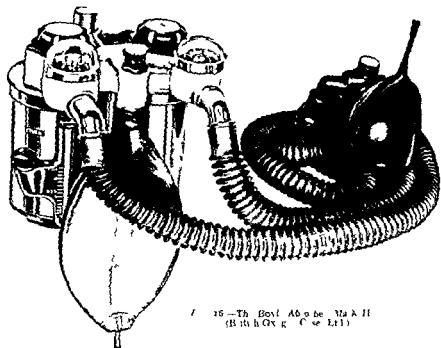


Fig. 16—The Boyl Air-o-be Mark II  
(British Oxygen Co. Ltd.)

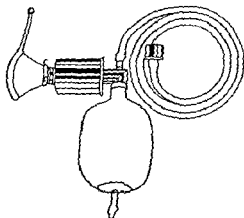


Fig. 17—Cope to-and-fro carbon dioxide absorber for infants.  
(British Oxygen Co. Ltd.)

centre and on the peripheral vessel wall. This latter action is not seen with ether\*.

At about the point of respiratory arrest when alveolar concentration of cyclopropane is around 40 per cent and independently of hypoxia and hypercapnia cardiac arrhythmias may occur and are mainly of ventricular origin. These may be due to a reflex action through a centre in the hypothalamus which sends impulses along the cardiac accelerator sympathetic nerves. These arrhythmias are usually abolished when the alveolar concentration is increased to 60-70 per cent. There is thus a zone of arrhythmias with normal heart beats both above and below the zone. Arrhythmias may take the form of bradycardia, multifocal ventricular tachycardia, pulsus alternans, A-V block or ventricular extrasystoles. Extrasystoles often alternate with a normal beat producing bigeminal pulse. A-V nodal rhythm also occurs but cannot be diagnosed by the finger or stethoscope. Arrhythmias are also stated to arise because of impulses which originate in the mesentery of the terminal ileum and proceed via the coeliac plexus splanchnic nerves and spinal cord to a centre in the brain; the efferent pathway involved is via sympathetic cardiac nerves. Bilateral thoracolumbar sympathectomy and splanchnicectomy remove the effect of adrenaline on the heart during cyclopropane anaesthesia†. Vagotomy has no effect on these arrhythmias. Hypoxia accentuates them as it causes adrenaline to be liberated into blood stream; deep cyclopropane anaesthesia and hypoxia are a dangerous combination. Total heart block and ventricular fibrillation may occur. Premedication with nembutal or induction with intravenous barbiturates lessens the frequency of arrhythmias possibly because they depress the suprarenals and lessen the amount of adrenaline in the circulation. Bradycardia, arrhythmia and tachycardia are usually regarded as indications for reducing the concentration of cyclopropane. Serious cardiac effects are not likely to precede respiratory arrest in the absence of rapid induction with high concentrations of the gas. If the concentration is increased after the onset of bradycardia the heart rate will probably become slower or arrhythmic or very fast and finally there is the danger of ventricular fibrillation. A heart rate slowed to 50 per minute may be regarded as a normal response. Arrhythmias are more frequent in ascent than in descent to deep anaesthesia. Atropine has no effect on while the addition of ether vapour lessens the frequency of arrhythmias. It is the opinion of Johnstone‡ that ventricular arrhythmias due to cyclopropane are often caused by carbon dioxide retention and can be abolished by efficient carbon dioxide elimination.

Beaconsfield, P. and Messent D. *Anesthesiology* 1955 18 429.

† Stutzman, J. W., Murphy Q., Allen, C. R. and Meek, W. J., *Anesthesiology* 1947 8 579.

‡ Johnstone, M., *Brit. Heart J.* 1950 12 239.

*Cyclopropane continued*

**Pharmacology and Effects**—It is the most potent of the anæsthetic gases so that it is always possible to employ at least 20 per cent of oxygen with it. Undergoes no chemical change in the body. Absorbed from the alveoli and excreted into the alveoli the blood and skin part with small volumes to the outside air.

Resembles ether in its anæsthetic effects but is less irritating to the respiratory tract more depressing to the respiratory centre and is more likely to cause cardiac arrhythmias in high concentrations. A concentration of 4 per cent will produce analgesia. 8 per cent light anæsthesia. 20 per cent to 25 per cent moderate anæsthesia. 40 per cent respiratory failure. Major part eliminated unaltered by lungs in 10 minutes but complete de saturation takes much longer.

**CENTRAL NERVOUS SYSTEM**—It is a potent and complete anæsthetic agent which stimulates the parasympathetic system. In concentrations up to 5 per cent it is a good analgesic agent.

**RESPIRATORY SYSTEM**—Non irritating to mucous membranes in concentrations under 50 per cent but reflex laryngeal spasm may result with higher concentrations partly because gas is vagomimetic. Apnoea produced when concentration approaches 40-45 per cent depending on premedication etc. Obstruction of the lower respiratory tract by bronchospasm may occur as it is a bronchoconstrictor. It does not stimulate the respiratory centre.

**BIOCHEMICAL CHANGE**—No evidence of interference with liver function. transient increase in blood sugar may occur. The kidneys do not secrete during the anæsthesia a neurogenic effect but a compensatory polyuria follows it.

**ALIMENTARY CANAL**—In light planes gut is contracted and peristalsis is present. these conditions are absent in deeper planes of anæsthesia. After anæsthesia the gut rapidly regains its normal tone. Nausea and vomiting are rarely prolonged.

**UTERUS**—Contractions not inhibited in light planes but abolished in deep planes. Blood concentration in foetus equals that of mother after 15 minutes.

**HÆMOPOIETIC SYSTEM**—Spleen increased in size. Leucocytosis at its maximum after 8 hours.

**MUSCLES**—Skeletal muscles easily relaxed but anterior abdominal muscles are capricious in this respect. Diaphragmatic movements not exaggerated in third plane anæsthesia as they some times are with ether at this level. Masseters easily relaxed. Tone of muscles tends to remain although reflex response to stimuli of trauma is abolished. Iris not much altered in light planes but pupils dilated in very deep anæsthesia.

**CARDIOVASCULAR SYSTEM**—In Planes 1 and 2 the amount of bleeding varies inversely with the depth of anæsthesia. in Planes 3 and 4 blood loss depends on whether or not a respiratory acidosis is prevented by proper ventilation. In Plane 2 cyclopropane causes more bleeding than ether. When vaso dilatation occurs the effect is in two parts on the vasomotor

(Nickerson and Goodman\*) and so also are procaine its constituent diethylaminoethanol and procaine amide pethidine the dihydrogenated ergot alkaloids and diethyl ether

**Views of Guedel**—In a masterly paper † Guedel advances unorthodox views based on a vast experience. He deals with cardiac arrhythmia abdominal relaxation and technique.

He finds arrhythmias more frequent in neurotic and sthenic adults between puberty and fifty. In the arrhythmic range anything may happen at any time. A pulse-rate greater than 200 is regarded as being due to ventricular tachycardia. Unusual to enter or leave a tachycardia without passing through a zone of arrhythmia. Arrhythmias occur under chloroform and ether and with these agents indicate circulatory depression. cyclopropane arrhythmias have no such significance. Circulatory efficiency is gauged by noting the arteriolar capillary refill time (A.C.R.). This is time taken for colour to return to a given area of skin after pressure has made it ischemic. The A.C.R. is reduced in hæmorrhage and shock. If colour is good and A.C.R. is within normal limits for the patient then the circulation is efficient however bizarre the pulse may be. He rejects the view that increasing concentration of cyclopropane in the blood displaces the pacemaker progressively downwards from the sino auricular to the auriculo ventricular node thence through the bundle of His and the branch bundles to the automatic conductive tissue and ventricular fibrillation reason that increasing the concentration usually leads to a regular pulse.

Muscular relaxation with cyclopropane can equal that of spinal analgesia if the concentration is raised high enough. This applies to all patients and pushing of the gas is not contra indicated by pulse abnormalities.

Serious cardiac abnormalities are complete heart block and ventricular fibrillation and as they are most likely to occur during the arrhythmic range this is gone through as quickly as possible. Induction is rapid a high concentration of gas being used which soon results in apnoea. From this point the breathing is controlled more and more cyclopropane being given until the arrhythmic range is left behind thus serious effects are minimized.

**Views of Waters.**—R. M. Waters who introduced cyclopropane into clinical practice in 1945 summed up his opinions of the gas and its uses\*.

He prefers the premedication to be given in small doses. He regards controlled respiration as being used too frequently. It should only be used to help the surgeon. Arrhythmia also should be avoided by decreasing the cyclopropane concentration if necessary. Induction should be by  $N_2O$  and  $O_2$  with rapid addition of cyclopropane to the mixture. In normal patients

\* Nickerson, M., and Goodman, L. S. *J. Pharmacol.* 1947 63 167

† Guedel, A. E., *Anesthesiology* 1940 13.

Waters R. M. *Surgery* 1945 18 26



Pharmacology and Effects—Cardiovascular System *continued*

Debatable point as to whether cyclopropane has a depressing effect on the myocardium in the absence of hypoxia. May or may not be comparable with chloroform in this respect. Some observers hold that dilatation and deficient contraction are present and even in concentrations of 15 per cent in the inspired mixture reduction in cardiac reserve may occur. Myocardial dilatation has recently been shown to be less with cyclopropane than with ether\*. Recent work† would suggest that the cardiac output and heart rate are reduced in both light and deep cyclopropane anaesthesia the former effect may be due to the latter effect and to elevation in total peripheral resistance. The drug has a pressor action on the systemic and pulmonary circulations.

Adrenaline further sensitizes the cardiac automatic conducting tissue to the presence of cyclopropane and all concentrations of cyclopropane sensitize the myocardium to circulating adrenaline. Adrenaline given intrathecally however to prolong the effect of spinal analgesia is absorbed very slowly and is compatible with general cyclopropane anaesthesia. Methoxamine (vasoxyl) and neosynephrin do not share in this effect. Mephenteramine (mephin) and ephedrine do not cause ventricular fibrillation when injected into patients under the influence of cyclopropane but have instead a protective effect against this arrhythmia. They do not prevent the tachycardia or multifocal extrasystoles caused by adrenaline during cyclopropane anaesthesia. Pituitrin is contra indicated as it may cause constriction of the coronary arteries and reflex bradycardia through vagal stimulation. Several deaths have been reported as due to ventricular fibrillation caused by the combination of cyclopropane and adrenaline. The danger is greater in deep than in light anaesthesia. Manipulation of the oesophageal and gastric branches of the vagus under cyclopropane anaesthesia may cause significant electrocardiographic changes‡.

Intracardiac or intravenous novocain may possibly prevent the ventricular fibrillation sometimes produced by these two agents. 10 ml of 1-2 per cent solution can be injected to try to regularize an abnormal rhythm or during cyclopropane anaesthesia 0.2 per cent procaine can be run into the patient's veins as a measure of protection against arrhythmia. Quinidine sulphate or lactate 100-200 mg intravenously combats serious cardiac arrhythmias and can be given as premedication when it lasts for two to four hours it may need to be repeated.

Dibenamine (N,N dibenzyl  $\beta$  chloroethylamine) and priscol (2 benzyl 4,5 imidazoline hydrochloride) are reported to provide good protection against irregularities due to cyclopropane.

Fisher C. W. Bennett L. L. and Allahwala A. *Anaesthesia* 1951 12 19

† Tsung Han Li, and Euston B. *Ibid.* 1957 18 15

‡ Freeman, A. G. *Lancet* 1951 1 926

time to time to compensate for leaks and waste of gas through the skin and wound. Some workers like to keep a constant flow of 30 c.c. per minute during the first half hour of anaesthesia. The absorber is cut back into circuit after anaesthesia is induced. Nitrogen collects in the breathing bag from the tissues. This serves to prevent post-operative atelectasis as it forms a slowly absorbable supporting gas. Premedication with respiratory depressants should be minimal. Should the tidal exchange become inadequate the absorber can be temporarily cut out of circuit or pressure can be exerted on the breathing bag during inspiration. A finger should be kept on the pulse so that the gas concentration can be reduced should cardiac changes occur. Should the patient require a pharyngeal or intratracheal airway the gas mixture should be retained in the bag when possible when the mask is removed from the face. Blind intubation is relatively difficult because of the shallow breathing but the use of the laryngoscope is relatively easy because of the relaxation of the jaw and throat muscles.

- If relaxation is not sufficient by the time respiration is becoming depressed an intravenous injection of relaxant is given. If the heart becomes irregular it is safer to add a little ether vapour than to push further cyclopropane into the tissues. If difficulty with relaxation is foreseen then some form of regional block should be used after induction of anaesthesia or a relaxant may be used. Light cyclopropane anaesthesia together with the use of a muscle relaxant forms a useful and popular technique. The addition of a little ether vapour aids bronchodilatation.

Towards the end of the operation air is gradually introduced into the circuit to prevent changes due to the sudden cessation of breathing of a high oxygen atmosphere. Many workers routinely add ether vapour to the inhaled gases after induction thereby avoiding arrhythmia and facilitating relaxation.

**Sequelæ** —Owing to the slight upset of the body chemistry patients are usually sicker after cyclopropane than after a comparable ether anaesthesia. Similarly they are less sick. Circulatory disturbances are more frequent after this agent than after ether e.g. tachycardia or arrhythmia persisting into the post-operative period. Respiratory morbidity less after upper abdominal operations under cyclopropane than after either ether or spinal analgesia.

**Cyclopropane Shock** —A type of circulatory collapse after the return of the patient to the ward is sometimes seen after deep anaesthesia. It is accompanied by slow pulse unlike surgical shock. Dripps\* postulates that one cause may be the sudden return to normal of a blood-carbon dioxide tension which has been high for some time the supposition being that during cyclopropane anaesthesia the shallow breathing is accompanied by a raised blood carbon-dioxide level which maintains the blood pressure somewhat above normal. It is treated by intravenous fluids. After long operations under cyclopropane anaesthesia some workers inject a blood pressure raising drug e.g. methoxamine.

*Views of Waters continued*

the oxygen tension in the mixture should not be more than 20 per cent lest accumulation of carbon dioxide resulting from hypoventilation should be overlooked

He regards cyclopropane as being a very useful anaesthetic with rapid control of depth of anaesthesia impossible with any other inhalation agent. He points out the very narrow margin between abdominal relaxation and respiratory paralysis

**Views of Griffith \***—H. R. Griffith writing of his experiences of 20 000 cyclopropane anaesthetics states that the gas should be treated with a healthy respect but he has had only one death in the series. His advice for keeping out of trouble (1) Maintain a free airway (2) Ensure proper pulmonary ventilation by assisted or controlled respiration (3) Aim for a smooth level of anaesthesia avoiding too strong a vapour tension. He uses the drug for patients with decompensated heart disease and with irregular hearts. Arrhythmia when it occurs is ignored with the exception of severe tachycardia (over 140) which may be the precursor of ventricular fibrillation and must be abolished by the use of a lowered tension of the gas the addition of a little ether vapour or intravenous procaine. Laryngeal spasm may occasionally be seen and usually responds to intravenous suxamethonium if oxygen cannot be insufflated the cords must be parted with a gum-elastic or other firm endotracheal tube. For relaxation during the operation a muscle relaxant is used.

In the present state of our knowledge it would seem wise to take the more conservative view as to the potential dangers of cyclopropane and to regard gross alterations in the pulse as indications for lowering the concentration of the gas.

**Technique of Administration**—Must be given in a closed-circuit machine with carbon dioxide absorption owing to high cost of gas—about 2s 9d per gallon. Machine tested for leaks by filling reservoir bag occluding gas exit and applying pressure to bag. Bag now half filled with either oxygen or air and face piece applied to face forming an airtight joint. Alternatively induction may be by minimal thiopentone. Cyclopropane added to circuit at rate of 250 to 400 c.c. per minute and basal oxygen added at about similar rate. Induction is speeded by building up carbon dioxide with the absorber out of circuit. Laryngeal spasm is not very uncommon during induction of anaesthesia. Unconsciousness comes on in less than a minute. Anaesthesia usually established in 5 minutes delirium being uncommon. When required plane is reached cyclopropane is reduced. Because of the large carotid blood supply to the brain during induction the cerebral tissue contains more cyclopropane than the rest of the body tissues. Redistribution gradually takes place until equilibrium is attained thus extra gas must be added in the first 10–20 minutes to keep the cerebral concentration at an adequate level. As the anaesthesia progresses more cyclopropane is added from

time to time to compensate for leaks and waste of gas through the skin and wound. Some workers like to keep a constant flow of 50 c.c. per minute during the first half hour of anaesthesia. The absorber is cut back into circuit after anaesthesia is induced. Nitrogen collects in the breathing bag from the tissues; this serves to prevent post-operative atelectasis as it forms a slowly absorbable supporting gas. Premedication with respiratory depressants should be minimal. Should the tidal exchange become inadequate the absorber can be temporarily cut out of circuit or pressure can be exerted on the breathing bag during inspiration. A finger should be kept on the pulse so that the gas concentration can be reduced should cardiac changes occur. Should the patient require a pharyngeal or intratracheal airway the gas mixture should be retained in the bag when possible when the mask is removed from the face. Blind intubation is relatively difficult because of the shallow breathing but the use of the laryngoscope is relatively easy because of the relaxation of the jaw and throat muscles.

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**Sequelæ—Cyclopropane Shock continued**

20 mg intramuscularly before the patient leaves the theatre. If this theory is correct the condition should not occur if the patient is properly ventilated during the operation.\*

*Atelectasis*—Soon after operation has been reported

*Emergence Delirium*—A condition of muscular activity and restlessness during emergence from anaesthesia is sometimes seen in the type of patient mentioned by Guedel as being specially liable to cardiac arrhythmias. A little ether given towards the end of the operation may prevent or lessen this. Injection of subemetic doses of apomorphine may also be beneficial— $\frac{1}{3}$  gr dissolved in 10 ml of saline 1 ml being injected at frequent intervals until the turmoil ceases. The intramuscular injection of 5 ml of paraldehyde to which has been added a small quantity of hyaluronidase has also a good sedative effect.

**Indications**—Useful in any case requiring smooth breathing with minimal after effects and minimal respiratory irritation. Very suitable for cases of shock and recent hæmorrhage, eclampsia, acute respiratory infection, cardiac decompensation, thoracic surgery. When given with an equal volume of oxygen cyclopropane can be relatively safely used to induce anaesthesia in patients with small amounts of material in their stomachs provided that the table is inclined with the head up and that a cuffed endotracheal tube is inserted quickly after a suitable dose of relaxant. The fall in blood pressure in patients so dealt with is less than that seen after induction in the head up position with an intravenous thiobarbiturate. (This method of induction is not for the beginner.)

J G Bourne† has designed a portable piece of apparatus for the administration of cyclopropane to patients in the dental chair which consists of a special face mask, a 6 litre breathing bag and a rapid means of filling the bag from two small sparklet miniature compressed gas containers filled with cyclopropane and oxygen nitrogen (50/50) mixture. The mixture of gases is non-flammable. For induction of anaesthesia the patient breathes in and out of the bag for 12–20 respirations, the face piece is taken away and the dentist has two or three minutes to complete his extractions. Prolongation of anaesthesia should be by nitrous oxide and oxygen given through a nasal inhaler. This excellent method which is technically easy is unfortunately followed by nausea and vomiting in some patients but this can be benefited by sublingual administration of a tablet of hyoscine (— 1.5 gr) half an hour before anaesthesia. The method can be warmly recommended.

**Contra indications—**

- 1 During operations involving use of diathermy or cautery unless circuit is really leakproof. This is a difficult achievement when controlled breathing with its increased pressure is used.

- 2 Some patients do not settle down well with this agent. They may show pallor early on, may develop laryngospasm and bronchospasm, or show cardiac arrhythmias at a light plane of anaesthesia. In such cases cyclopropane should be abandoned.
- 3 When adrenaline is to be used. Nevertheless thousands of operations have been successfully performed in which adrenaline has been injected into patients under cyclopropane. If a nor-adrenaline drip is in use along with spinal analgesia cyclopropane may probably be used as a supplementary anaesthetic provided that the spinal analgesic reaches to T5 with block of the splanchnic nerves. This high spinal block breaks the reflex arc concerned with the production of cardiac arrhythmias of a serious nature which are said to originate in the mesentery.
- 4 For the unskilled anaesthetist cyclopropane is contra-indicated.
- 5 In very young children unless specially designed apparatus is used. Otherwise the amount of dead space will lead to carbon dioxide accumulation while resistance to breathing may exhaust the child and produce jerky respiration.

**Safety**—Its cardiac effects make it less safe than ether. Sudden deaths have occurred, presumably due to ventricular fibrillation, not all of them in deep anaesthesia.

**Note for Beginners**—First become familiar with the closed circuit using ether. Then commence the use of cyclopropane on minor cases with only atropine as premedication. For abdominal cases always have the patient intubated so that when respiratory depression occurs interchange of gases can be carried out without the worry of having to maintain a difficult airway. Beware of tachycardia over 140 per minute.

## CONTROLLED RESPIRATION

This was introduced by Guedel and Treweek in 1934 using ether. Starting off in Stage III Plane 2 they quickly increased the ether concentration and produced hyperventilation by bag pressure in a closed circuit with the soda lime canister in operation. They thus caused a raised threshold of the respiratory centre together with a lowering of the stimulant to respiration—carbon dioxide and apnoea followed in about four minutes—the so-called ether apnoea. Waters in 1936 first used the term controlled respiration, but perhaps controlled apnoea would be a better term. It is the temporary loss of respiratory drive due to either central or peripheral depression. Controlled apnoea, while formerly produced by cyclopropane, is now usually obtained by the use of muscle relaxants. Crafoord reported on his spiro-pulsator in 1940 and in the following year Nosworthy's classic paper appeared\*. Apnoea can be produced during anaesthesia by—

- 1 Raising the threshold of the respiratory centre as by barbiturates, opiates or cyclopropane, or a combination of these—a pharmacological method. The blood carbon-dioxide no longer stimulates

**Controlled Respiration continued**

respiration All volatile agents if pushed will cause apnoea in this way

- 2 Depleting the physiological stimulus to respiration carbon dioxide by hyperventilation When stimulus falls below threshold there is respiratory depression This is a physiological mechanism In addition a rich oxygen supply removes the respiratory drive originating in the aortic and carotid bodies A combination of the two methods may be employed
- 3 Voluntary respiration is abolished by the use of curare or one of its substitutes by paralysing the respiratory muscles while anaesthesia is maintained by a combination of nitrous oxide oxygen and thiopentone and pethidine

A brief apnoeic period as is sometimes required in radiological procedures such as bronchography pyelography or cholangiography can be produced by activation of the Hering Breuer reflex brought about by continued pressure inflation of the lungs at the end of inspiration if the anaesthetic in use has not inactivated this reflex response to inflation of the lung Such agents as thiopentone gas and oxygen and light gas-oxygen-ether do not depress these reflexes Deep ether cyclopropane and chloroform on the other hand inactivate these Hering Breuer reflexes

Apnoea may follow a sudden rise in blood pressure as by intravenous adrenaline an effect mediated through the aortic and carotid sinuses

**Assisted Breathing**—Manually assisted inspiration implies some rhythmic activity of the respiratory centre and the respiratory muscles Pressure on the reservoir bag should be synchronous with inspiration while it is completely withdrawn to allow full passive expiration

**Intermittent Positive Pressure Respirator (IPPR)**—Includes both controlled and assisted respiration

**Physiological Changes associated with Controlled and Assisted Breathing**—In positive pressure breathing inspiratory intrapulmonary and intrapleural pressures are reversed from what they are during normal breathing—i.e. they become positive instead of negative

**Respiratory alkalosis** may be caused This tends to increase the affinity of haemoglobin for oxygen to cause cerebral vasoconstriction and to decrease cardiac output but is seldom clinically important Over ventilation raises the pain threshold probably due to cerebral hypoxia (from cerebral vasoconstriction) \*

*The central venous pressure is increased and with this the systemic blood pressure and the cardiac output are decreased The rise in central venous pressure follows the displacement of blood from the lungs into the systemic circulation by interfering with*

the entrance of blood into the chest. These circulatory effects are made less by allowing for prolonged expiration. The interference with the venous return does not seem harmful in practice. Oxygen consumption is reduced because of the easing of the patient's muscular effort.

It may be that the use of controlled apnoea reduces the necessary doses of relaxant and narcotic.\*

**Technique**—The circuit must be able to withstand the positive pressure applied to the breathing bag without leaking unduly. With an endotracheal tube under the mask the stomach may become distended with air. A Ryle's tube or an oesophageal tube will prevent this. The former can be brought under the mask and aspirated occasionally or a balloon can be fixed to it to distend the oesophagus as suggested by Macintosh. The junction between the machine and the patient also must be leak proof and may be arranged as follows—

- Use of a well fitting mask and harness
- Use of an endotracheal tube with inflated cuff or gauze pack
- Use of an endotracheal tube with the lower jaw bandaged backwards to occlude the pharynx (Pinson)
- Use of gauze swabs or Thornton's mouth prop to build up the cheeks of an edentulous patient to enable an airtight joint to be made between mask and face. Moistened 2 in bandages in the cheeks are also useful

**1. USING MUSCLE RELAXANTS**—Induction is by intravenous thiopentone and maintenance is by nitrous oxide and at least 30 per cent of oxygen. When the production of apnoea is desired the relaxant is injected intravenously in divided doses and the patient is hyperventilated until apnoea is produced. Further doses are given as may be necessary. It is frequently desirable to stop short of frank apnoea in which case assisted breathing is required to ensure adequate gaseous interchange. The reservoir bag is gently compressed with each of the patient's shallow inspirations. The necessity is again emphasized of seeing that breathing of normal depth has returned before the patient is sent from the theatre. If necessary neostigmine should be used to counteract the curare effect preceded by atropine. It must be used only after careful consideration if the respiratory depression is due to suxamethonium compounds.

The pressure required to inflate the lungs varies with a number of factors including (a) The depth of anaesthesia (b) Whether a relaxant has been used or not (c) Whether the chest wall is open or closed (d) The pathology of the lungs (e) The patency of the airways. Rupture of the lung and its sequelae pneumothorax, mediastinal emphysema, pulmonary interstitial emphysema and subcutaneous emphysema are unlikely if due care is taken. Never should a pressure of 20 mm Hg be exceeded—even though during a severe bout of coughing an interbronchial pressure of 75 mm Hg has been recorded.



## Technique continued

- 2 USING CYCLOPROPANE —The respiratory centre is depressed by *morphine* premedication and a *thiopentone* induction. Depletion of carbon dioxide can be carried out by gently compressing the bag with each inspiration so that the expired carbon dioxide is absorbed in the soda lime. This hyperventilation soon produces a blood carbon-dioxide level too low to stimulate the respiratory centre. Tidal exchange is then maintained by compression of the breathing bag. Bag pressure whatever combination of drugs is being used should be applied 15–20 times each minute at a pressure not exceeding 10 to 12 mm Hg the duration of expiration being twice that of inspiration so as to ensure complete elimination of carbon dioxide. This also minimizes the circulatory effects of positive intrapulmonary pressure. The more cyclopropane that is added the sooner will apnoea appear. Thus breathing is directly controlled by the anaesthetist both as to rate and depth. Periods of complete apnoea with cessation of all respiratory movement can be allowed to help the surgeon during a specially difficult stage of the operation so long as serious anoxia does not occur. This is very useful in thoracic surgery.

Towards the end of the operation the bag is emptied and refilled with 50 per cent nitrous oxide-oxygen or air and the absorber cut out of circuit. Tidal exchange is kept up as before and the procedure repeated each minute. When active breathing recommences the absorber is again employed and the patient finishes the operation breathing normally. If the early breaths are shallow they must be assisted by gentle bag pressure. If cyclopropane has been pushed normal respiration may be delayed for 10–15 minutes after the end of the operation. The anaesthetist must not leave the patient until reasonably normal breathing has been re established for a minute or two lest apnoea should reappear and cause death from anoxia. Real danger of subsequent apnoea exists if a patient is suddenly removed from a circuit containing gases abnormally high in carbon dioxide tension.

## Assessment of Depth of Anaesthesia with Controlled Apnoea —

(1) Anaesthesia is light if respiration rapidly returns after a little carbon dioxide is allowed to accumulate. (2) The ease with which apnoea develops during hyperventilation through soda lime is an index of depth and of respiratory centre depression using any 100 per cent anaesthetic agent. (3) Resistance to inflation decreases with increased depth of anaesthesia whether chest is open or closed. (4) Light anaesthesia is suggested if the chest but not the abdomen moves on squeezing the bag. Good relaxation is only present when both chest and abdomen move on bag pressure. (5) Under thiopentone the duration of apnoea from activation of the Hering Breuer reflexes by inflation of the lungs is longer the greater the depth of anaesthesia (this is before the onset of controlled apnoea). (6) The eye signs. (7) The presence or absence of swallowing. (8) Traction reflexes are more likely to

break through apnoea due to overventilation than through that due to relaxants (9) When cyclopropane is being used the smell of the mixture should indicate its concentration (10) Knowledge of the amounts of drugs used and length of time of anaesthesia (11) Using gas-oxygen-ether and perhaps cyclopropane but not gas-oxygen thiopentone when respiratory movements are present if the reservoir bag is squeezed at the end of inspiration so as to prevent expiration a definite respiratory pause occurs in Planes 1-2 being longer the lighter the plane of anaesthesia and lasting for several respiratory cycles This reflex disappears before intercostal activity ceases and its presence is a rough index of lightness \*

When employing the technique of controlled apnoea it is important for the anaesthetist to keep in mind (a) The blood carbon dioxide tension (b) The degree of depression of the respiratory centre (c) The amount of muscular paralysis

**Advantages**—When muscle relaxants are used controlled breathing is very often necessary in order to provide a proper interchange of gases In thoracic surgery controlled or assisted respiration prevents paradoxical breathing i.e. prevents inflation of the collapsed lung during expiration and deflation during inspiration with the inefficient interchange of gases so resulting It also prevents mediastinal flap in those cases with a mobile mediastinum and makes breathing more efficient when the mediastinum is rigid The quiescence of the diaphragm produced by controlled respiration aids the work of the surgeon when he is near this muscle either in the upper abdomen or in the thorax It may also reduce the amount of thiopentone and relaxant required during the operation thus contributing to the speedy recovery of consciousness and muscle tone † Assisted (or controlled) breathing is advisable at some time in almost all cases of inhalation anaesthesia requiring muscular relaxation (except when ether is the main agent) as it decreases the incidence of hypoxia and respiratory acidosis

**Disadvantages**—The respiratory signs of anaesthesia are abolished Pressure changes in the chest may adversely influence the circulation and impede the venous return to the heart

**Machines for Artificial Respiration**—These may be operated by hand by electricity or by pressure from a cylinder of compressed gas A good machine should provide for a phase of rapid inflation with short peak pressure followed by a prolonged and complete expiratory phase at atmospheric pressure In some models exhalation can be helped by a phase of negative pressure A machine should deliver a controllable gas flow and volume and the change from inspiration to expiration should be preselected Some types are designed so that they are triggered by the change which occurs between inspiration and expiration however slight this may be others again are worked by a pump with a safety valve to prevent the development of too high an intrathoracic pressure

*Mode of Action continued*

b There is a great difference in effect following the inhalation of 20 per cent oxygen with 80 per cent nitrous oxide and following 20 per cent oxygen-80 per cent nitrogen (air)

c Respiratory arrest is produced much quicker after inhalation of pure nitrous oxide than after inhalation of pure nitrogen

Nevertheless many of the clinical signs of well administered nitrous oxide-oxygen anæsthesia are due to the asphyxial element, this being necessary owing to weak anæsthetic properties of nitrous oxide

The normal amount of hæmoglobin is 15 g in 100 ml of blood. As each gramme can combine with 1.34 ml of oxygen the 15 g in 100 ml will combine with 20 ml of oxygen. In addition the 100 ml of plasma will dissolve about 0.24 to 0.3 ml of oxygen and carry it in solution

100 ml of blood will dissolve (in its plasma) 45 ml of nitrous oxide. The amount of gases which can be carried other factors being equal by a given volume of blood in simple solution depends on the proportions or tensions of these gases in contact with the blood. Therefore the proportions inhaled determine the tensions of those gases held in solution. The amount of nitrous oxide absorbed from the lungs is dependent on its concentration or partial pressure. It follows that the tension or proportion governs the depth of anæsthesia.

There are some recent reports which suggest that the administration of nitrous oxide over periods of many hours as in the treatment of tetanus or bulbar poliomyelitis may lead to severe bone marrow aplasia and fatal neutropenia.\*

**Stages of Anæsthesia**—The Guedel classification does not apply to nitrous-oxide-oxygen anæsthesia with full oxygenation. With full oxygenation greater depth than a point corresponding to Plane 1 Stage III is impossible in healthy patients. When the factor of hypoxia is added greater depth is obtainable but the signs are then partly those of asphyxia.

Thus in clinical nitrous-oxide-oxygen anæsthesia it is possible to recognize—

Stage I—analgesia

Stage II—delirium

Stage III—surgical anæsthesia

Stage IV—bulbar paralysis due to acute asphyxia

It is important to know if the patient in surgical anæsthesia is (1) Too light (2) Adequately anæsthetized (3) Too deep

Opinions differ as to the degree of oxygen lack permissible. Moderate hypoxia in fit patients for short periods is probably not harmful. The present trend is towards the addition of small amounts of supplementary anæsthetics e.g. thiopentone, vinesthene, trilete etc. rather than pushing pure nitrous-oxide-oxygen.

**Signs of Anæsthesia**—Satisfactory nitrous-oxide-oxygen anæsthesia for more than a few minutes requires some premedication and this will tend to mask signs of anæsthesia.

Most of the signs of nitrous-oxide-oxygen anæsthesia depend on the reaction of muscles. The depth of anæsthesia depends on the relative proportions of nitrous oxide and oxygen while a consideration of the signs is the chief guide to the proportions of gases the patient requires from time to time.

**INFLUENCE OF PREMEDICATION**—Atropine may mask the slow bounding pulse of oxygen want. Pentobarbitone may mask many of the signs of depth. Morphine and pethidine do not mask the signs of nitrous-oxide-oxygen anæsthesia provided they have been given at least one hour beforehand. Morphine scopolamine is less misleading than morphine atropine.

- 1 **ANALGESIA—STAGE I**—During induction this is passed through very quickly. Sustained analgesia is easy to produce and is most useful in the preparation of dental cavities in obstetrics and in minor surgery e.g. changing painful dressings etc. It usually requires about 45 per cent nitrous oxide in air i.e. 11 per cent of oxygen or 23 per cent to 33 per cent oxygen with nitrous oxide. Subjectively there is a feeling of euphoria, bodily warmth and slight vertigo while the attention is strongly focused on the sense of hearing. Pain sense is progressively depressed but intense stimuli such as skin incisions, removal of dental pulp etc. are not obtunded. Premedication or insufficient oxygen may produce unconsciousness or lack of control.
- 2 **DELIRIUM—STAGE II**—This is almost completely confined to (a) The frightened (b) The tough and sturdy (c) The alcoholic who will seldom take gas and oxygen smoothly.

The apparent stimulation is due to paralysis of the higher centres of inhibition and can often be minimized by premedication. This stage is often seen during ascent from deeper anæsthesia a fact which gave nitrous oxide the early name 'laughing gas'.

- 3 **SURGICAL ANÆSTHESIA—STAGE III**—In his book *Nitrous Oxide-Oxygen Anæsthesia* \* Clement separates this stage into—
  - a Light anæsthesia characterized by reflex movements often resistive and related to surgical stimuli.
  - b Normal anæsthesia with relative muscular relaxation.
  - c Deep anæsthesia with hypoxic rigidity of muscles and perhaps jactitation unrelated to surgical stimuli and serving no purpose.

**RESPIRATORY SIGNS**—These are the most useful signs of all but are only valid if a free airway is maintained at all times. Every breath must be seen or heard. Regular breathing marks the onset of surgical anæsthesia.

- a **LIGHT ANÆSTHESIA**—Rate is increased a reaction to oxygen lack. Rhythm may be altered by surgical stimuli. Apnoea may occur as breath holding—at the end of inspiration. Phonation may occur due to reflex laryngeal spasm from surgical stimuli.
- b **NORMAL ANÆSTHESIA**—Rate quicker than normal breathing. Rhythm regular inspiration and expiration being equal in duration. Phonation absent.

## Signs of Anæsthesia—Respiratory continued

- c* **DEEP ANÆSTHESIA**—Rate increased and later slows up becoming gasping (air hunger) Rhythm jerky and irregular Expiration prolonged with short jerky inspiration due to hypoxic spasm of diaphragm and intercostals Phonation may be present and is due to hypoxic spasm of larynx it may be preceded by furrowing of the tongue which can be seen in dental work

Apnoea may occur but it is at the end of expiration unlike the breath holding of light anæsthesia it is respiratory arrest The signs of deep anæsthesia are those of hypoxia They may be absent in anæmic or feeble patients who may just fade out if they become seriously hypoxic

*Respiratory Arrest*—This is the asphyxial Stage IV and is due either to spasm of the muscles of expiration or to paralysis of the respiratory centre—in either case due to hypoxia The heart will usually beat for a minute or two except in cases of grave myocardial disease Thus artificial respiration will usually revive the patient oxygen given under pressure being the best form of resuscitation

## MUSCULAR SIGNS—

- a* **LIGHT ANÆSTHESIA**—Movements reflex and resistive  
*b* **NORMAL ANÆSTHESIA**—Movements absent relative relaxation  
*c* **DEEP ANÆSTHESIA**—Rigidity twitching jactitation even opisthotonos Increased oxygen will abolish these movements With progressive asphyxia muscles become atonic including the larynx at the stage preceding death

**EYE SIGNS**—These are more useful when a patient has been stabilized in anæsthesia than during induction and light short anæsthesia Morphine and barbiturate may mask these signs

- a* **LIGHT ANÆSTHESIA**—Eyeballs move actively Eyes tightly closed and eyelid reflex present Pupils react to light and may be dilated due to emotional sympathetic stimulation  
*b* **NORMAL ANÆSTHESIA**—Eyeballs less active tending to stay in position of rest in centre of palpebral fissure Lids not tightly closed and eyelid reflex absent  
*c* **DEEP ANÆSTHESIA**—Eyeballs pulled strongly in one direction usually downwards through hypoxic spasm the stronger muscles winning Squint may occur Pupils enlarge becoming inactive to light—hypoxic spasm of dilator pupillæ In the asphyxial Stage IV—pupils are widely dilated

**THE COLOUR**—Cyanosis results when 3.3 g to 5 g or more of hæmoglobin in each 100 ml of blood is circulating uncombined with oxygen Some authors express it as an unsaturation of venous blood exceeding 11.4 vol per cent—the normal unsaturation of venous blood being 6 vol per cent Cyanosis is not clinically recognizable until the saturation of oxygen is reduced to between 85 and 75 per cent varying with different observers The amount of oxyhæmoglobin in circulation does not influence cyanosis As the blood of individuals

## NITROUS OXIDE-OXYGEN SIGN CHART OF THE THIRD STAGE OF ANESTHESIA

(McNesson)

	LIGHT ANESTHESIA	NORMAL ANESTHESIA	PROFOUND ANESTHESIA
	Due to too much oxygen in the mixture	Due to a properly balanced mixture of N <sub>2</sub> O-O <sub>2</sub>	Due to too little O <sub>2</sub> in the mixture or to partial obstruction of respiratory passages
Respiration	<ul style="list-style-type: none"> <li>a Superficial slow breathing often irregular</li> <li>b Prolonged inspiration Phonation due to reflexes of pain</li> <li>c Holding breath grunting</li> </ul>	<ul style="list-style-type: none"> <li>a Full machine like respirations Regular and faster than normal</li> <li>b Inspiration and expiration nearly equal</li> <li>c No phonation</li> <li>d Continuous uninterrupted respiration</li> </ul>	<ul style="list-style-type: none"> <li>a Irregular rhythm (sobby) usually slower than normal Spasmodic</li> <li>b Prolonged expiration</li> <li>c Phonation due to muscular spasm of vocal cords. Often crowing</li> <li>d Temporarily inefficient breathing Cessation of respiration usually from spasm of muscles of exhalation</li> </ul>
General muscles	<ul style="list-style-type: none"> <li>a Purposeful movements of rigid muscles</li> <li>b Facial expression of pain semi-consciousness</li> <li>c Nausea, very rarely</li> <li>d Reflex or purposeful resistance as result of trauma</li> </ul>	<ul style="list-style-type: none"> <li>a Immobile and relaxed</li> <li>b Exhibiting normal muscular tonus</li> <li>c Expression of normal sleep</li> <li>d Quiet Relaxed</li> </ul>	<ul style="list-style-type: none"> <li>a Clonic movements twitching or jerking in early minutes of induction often start in upper eyelids</li> <li>b Expression wild looking</li> <li>c Swallowing retching or vomiting common</li> <li>d Tetanic spasm in rigid body—opisthotonos some cases</li> </ul>
The eyes	<ul style="list-style-type: none"> <li>a Pupils large contract to light actively</li> <li>b Conjunctiva sensitive</li> <li>c Eyeballs roll quite rapidly</li> <li>d Eyelids resist opening wink when touched</li> </ul>	<ul style="list-style-type: none"> <li>a Pupils small or medium fixed</li> <li>b Conjunctiva insensitive to touch</li> <li>c Eyeballs fixed or slowly rolling</li> <li>d Lids often slightly open relaxed no winking</li> </ul>	<ul style="list-style-type: none"> <li>a Pupils fixed enlarging progressively and finally become irregular in outline</li> <li>b Conjunctiva insensitive</li> <li>c Eyeballs fixed in some position often downward or jerking</li> <li>d Eyelids stiff Often wide open</li> </ul>
Pulse rate	Accelerated	Usually slightly above normal	Rapid or very slow and sometimes irregular
Blood pressure	Normal	Normal	Sometimes increased slightly but usually decreased
Colour of skin and blood	<ul style="list-style-type: none"> <li>a Pink or no change normally</li> <li>b In anæmics no colour change</li> <li>c In plethorics, slight cyanosis</li> </ul>	<ul style="list-style-type: none"> <li>a Varies from pink to decided cyanotic tint</li> <li>b In anæmics, no colour change</li> <li>c In plethorics, considerable cyanosis</li> </ul>	<ul style="list-style-type: none"> <li>a Usually cyanotic</li> <li>b In anæmics, slight flush</li> <li>c In plethorics very blue</li> </ul>
Remedy	Decrease the percentage of oxygen in the mixture		Increase oxygen in the mixture or inflate lungs with pure oxygen 1 to 3 times

*Signs of Anæsthesia—Respiratory continued*

- c* **DEEP ANÆSTHESIA**—Rate increased and later slows up becoming gasping (air hunger) Rhythm jerky and irregular Expiration prolonged with short jerky inspiration due to hypoxic spasm of diaphragm and intercostals Phonation may be present and is due to hypoxic spasm of larynx it may be preceded by furrowing of the tongue which can be seen in dental work

Apnoea may occur but it is at the end of expiration unlike the breath holding of light anæsthesia it is respiratory arrest The signs of deep anæsthesia are those of hypoxia They may be absent in anæmic or feeble patients who may just fade out if they become seriously hypoxic

*Respiratory Arrest*—This is the asphyxial Stage IV and is due either to spasm of the muscles of expiration or to paralysis of the respiratory centre—in either case due to hypoxia The heart will usually beat for a minute or two except in cases of grave myocardial disease Thus artificial respiration will usually revive the patient oxygen given under pressure being the best form of resuscitation

**MUSCULAR SIGNS—**

- a* **LIGHT ANÆSTHESIA**—Movements reflex and resistive  
*b* **NORMAL ANÆSTHESIA**—Movements absent relative relaxation  
*c* **DEEP ANÆSTHESIA**—Rigidity twitching jactitation even opisthotonos Increased oxygen will abolish these movements With progressive asphyxia muscles become atonic including the larynx at the stage preceding death

**EYE SIGNS**—These are more useful when a patient has been stabilized in anæsthesia than during induction and light short anæsthesia Morphine and barbiturate may mask these signs

- a* **LIGHT ANÆSTHESIA**—Eyeballs move actively Eyes tightly closed and eyelid reflex present Pupils react to light and may be dilated due to emotional sympathetic stimulation  
*b* **NORMAL ANÆSTHESIA**—Eyeballs less active tending to stay in position of rest in centre of palpebral fissure Lids not tightly closed and eyelid reflex absent  
*c* **DEEP ANÆSTHESIA**—Eyeballs pulled strongly in one direction usually downwards through hypoxic spasm the stronger muscles winning Squint may occur Pupils enlarge becoming inactive to light—hypoxic spasm of dilator pupillæ In the asphyxial Stage IV—pupils are widely dilated

**THE COLOUR**—Cyanosis results when 3.3 g to 5 g or more of hæmoglobin in each 100 ml of blood is circulating uncombined with oxygen Some authors express it as an unsaturation of venous blood exceeding 11.4 vol per cent—the normal unsaturation of venous blood being 6 vol per cent Cyanosis is not clinically recognizable until the saturation of oxygen is reduced to between 85 and 75 per cent varying with different observers The amount of oxyhæmoglobin in circulation does not influence cyanosis As the blood of normal individuals

Prolonged hypoxia may in certain patients cause permanent damage to the cells of the cerebral cortex. Results may take the form of Jacksonianism, athetoid movements, idiocy or decorticate rigidity.

A full strong pulse indicates light or normal anaesthesia. An irregular pulse of poor volume or a rapid pulse is a sign of hypoxia.

**Diffusion Hypoxia**—Immediately the mask is removed from a patient who has for a longish period been breathing nitrous oxide with adequate oxygen, a high percentage of the expired volume may consist of nitrous oxide so that the outward diffusion of the gas lowers the partial pressure of oxygen in the alveoli. This gives rise to a condition known as diffusion hypoxia and it may be harmful in ill or handicapped patients. The remedy is to add oxygen to the inspired air during the quarter of an hour following the anaesthesia.

### Apparatus for Administration of Gas and Oxygen —

Dates of introduction of some machines: Hewitt 1898, Teter 1902,

McKesson 1910, Connell 1911, Foregger 1914, Boyle 1917.

Machines are (1) Continuous flow, (2) Intermittent flow, the gas flow being shut off during expiration.

#### 1. CONTINUOUS FLOW MACHINES —

*a* **THE BOYLE MACHINE (1915-18)**—The gases are delivered to the flow meter which usually measures nitrous oxide, oxygen, cyclopropane and carbon dioxide. The control valves are either on the gas cylinders or below the flow meter. Minimal amounts of vapour from the volatile anaesthetic can be added by passing the gases over the surface of the liquid; control is by a rotating tap. Two vaporizing bottles are usually provided. If further concentration of vapour is required, a plunger progressively depressed will give this by directing the gases so that they bubble through the liquid. Gases then pass into a Magill rebreathing attachment, a rubber bag (which can be cut out of the circuit), a length of wide bore corrugated tubing, an expiratory valve and a face piece. Reducing valves are attached to the cylinders to reduce the issuing pressure to about 5 to 7 lb. to the square inch.

To use the machine, the cylinder, after checking, are opened and nitrous oxide is allowed to flow at the rate of about 10 litres a minute. The mask is gradually lowered on to the patient's face and the expiratory valve is opened. Soon gas flow can be cut down to about 6 litres a minute and oxygen is admitted at the rate of 500 ml. to 2 litres a minute. The total gas flow should not be less than 8 litres a minute, otherwise carbon dioxide may accumulate in the circuit.

Modifications of the original Boyle machine of 1917, which was a two gas water sight feed, were —

1920 Addition of vaporizing bottle to flow meters

1926 Addition of second vaporizing bottle and by pass controls

1927 Addition of third water sight feed tube for carbon dioxide



**Signs of Anæsthesia—Colour continued**

contains 15 g of hæmoglobin in each 100 ml the onset of cyanosis leaves 10 g available for carrying oxygen. In anemia with a hæmoglobin estimation of 10 g per 100 ml the onset of cyanosis shows that only 5 g are available for oxygen transport which is thus reduced by half. In plethora with say 20 g per cent cyanosis still leaves 15 g for active duty.

Thus cyanosis must be correlated with the blood state of the patient. With severe anemia there will be no cyanosis until depression is grave while the full blooded patient is often cyanosed slightly even when breathing fresh air. In normal patients under gas and oxygen anæsthesia a moderate cyanosis may not be harmful in the absence of muscular signs of hypoxia. In the plethoric patient if anæsthesia with nitrous oxide-oxygen is possible it will necessitate considerable cyanosis.

The chief guide from colour changes occurs once a patient is stabilized when increasing cyanosis suggests increasing depth and vice versa.

**FACTORS MODIFYING CYANOSIS**—(1) Pigmentation of the skin either normal (e.g. race) or abnormal (e.g. in jaundice). (2) Thickness of epidermis. (3) Variation of density of capillary network in the observed skin area. (4) Temperature of skin—cold causes stagnation of circulation and consequent reduction of oxyhæmoglobin. (5) Increased venous pressure and high blood volume e.g. polycythæmia favours cyanosis. Cyanosis may thus not be very obvious in shock.

**PATHOLOGICAL CONDITIONS CAUSING CYANOSIS INDEPENDENT OF ANÆSTHESIA**—

## 1. Circulatory shunt from veins to arteries—

a. In Fallot's tetralogy (a narrow pulmonary artery patent interventricular septum, mouth of aorta overrides both left and right ventricles at the place where the septal defect lies, hypertrophied right ventricle and atrophied left ventricle).

## 2. In lung disease—

a. Lung consolidation collapse or œdema  
b. Tracheal obstruction  
c. Asthma  
d. Emphysema  
e. Azygos disease

## 3. In heart disease—

a. Mitral disease  
b. Congestive heart failure  
c. Pulmonary atresia or stenosis  
d. Tricuspid atresia

## 4. In alterations in blood pigment e.g. in sulphæmoglobinæmia and methæmoglobinæmia

weating due to sympathetic stimulation may occur in light anæsthesia.

Retching and vomiting usually indicate oxygen lack and deep anæsthesia. With ether these signs indicate lightness of anæsthesia.

oxygen that on the right measures nitrous oxide while the centre one is for carbon dioxide. A by pass lever is provided for the admission of five litres per minute of nitrous oxide which then does not bubble through the water in the bottle. This by pass is opened by raising

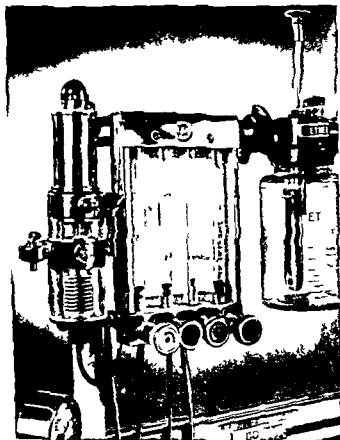


Fig 19 Rotameter Block Flowing the Rosin Audible Warning Device (British Oxygen Gases Ltd)

the lever from horizontal to vertical. This method is inaccurate and out of date but enables the anaesthetist to see the bubbles of gas actually being delivered to the patient.

- 11 *Coxeter's Dry Bobbin Flow meter.* Gas is led to the base of a small vertical glass tube with perforations surrounded

## Continuous flow Machines—Boyle's continued

- 1930 Addition of plunger device
- 1933 Dry bobbin type of flow meter displaced water sight feed
- 1937 Rotameters displaced dry bobbin flow meters

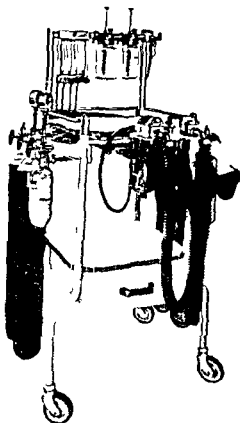


Fig. 18—Boyle's machine with rotameters (Model H) (British Oxygen Gases Ltd.)

## Flow meters—

1. *The Water Sight feed* A metal tube with five regularly spaced perforations is immersed in a water-containing bottle. Gas is fed to the tube and bubbles through the perforations; the number of bubbles indicating the rate of flow when read off on a chart provided with each machine. Three metal tubes are usually supplied as a unit in the same glass bottle—that on the left measures

series the liquid having the lower boiling point should be vaporized first. In this way recondensation of the vapour of the liquid from the first vaporizer is less likely to recondense in the second vaporizer. Thus the ether vaporizer should be next to the flowmeters and the trilene or chloroform vaporizer second in the serial gas train. If this is not done liquid trichloroethylene (boiling point  $87^{\circ}\text{C}$ ) for example can be

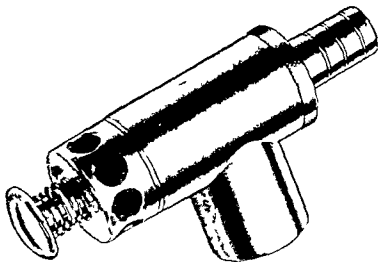


Fig. 1.—The Boyle Valve (Longworth Instrument Co. Ltd.)

recovered from the ether vaporizer (boiling point  $35^{\circ}\text{C}$ ). In the older types of Boyle machine the J tubes and plungers of the vaporizing bottles should be interchangeable.\*

The Bosun Visual Audible Warning Device (Fig. 19) warns the anaesthetist when the oxygen cylinder is empty by a reed type whistle and by a red light supplied by a 1.5 volt dry cell. These mechanisms are activated by gas from the nitrous oxide cylinder. The Bosun is a most useful addition to the anaesthetic machine.

## Continuous flow Machines—Boyle's continued

by an airtight glass cylinder. In the tube a bobbin is supported on the gas jet the height of the top of the bobbin indicating the rate of gas flow as measured by engraved numbers on the small tube. The gas escapes from the small tube through the perforations beneath the bobbin. Three vertical glass flow meters are enclosed in a single glass case so that nitrous oxide (on the right), oxygen (on the left) and carbon dioxide (in the centre) can be measured. This too is now outdated.

- iii *The Rotameter*. As a gas measuring device it was patented in Germany in 1908 by Karl Kuppers of Aachen and used in anaesthesia by Maximilian Neu in 1910. Magill

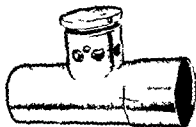


Fig. 10.—The Boyle and Salt's rotameter.  
(Boyle and Salt's Ltd.)

suggested its use independently in 1932 and used it a few years later. R. Salt developed it further in 1937. Gas is led to the base of a finely wrought glass tube slightly smaller on cross section at bottom than at top. The tube usually three or four in number are enclosed in a glass cylinder. A light metal float rides the gas jet and notches in its edge cause it to rotate. Height of top of float gives rate of flow, the gas escaping between the rim of the metal float and the walls of the glass tube. The glass tubes must be vertical. The calibrating of the glass tubes must take into effect both the density and the viscosity of the gases passing through them. Consequently a rotameter calibrated for carbon dioxide will not read true for cyclopropane because although their densities are similar (44/42) their viscosities are different (1.0/6). The type used today in most modern machines.

This is an accurate meter with an error of  $\pm 2$  per cent. The Boyle and similar machines are supplied with two vaporizing chambers for volatile anaesthetics. Where a common flow of gases passes through

- iv. The Salt valve (Fig. 21) Consists of a thin fibre disk on a knife-edge setting. Not spring loaded so opens without appreciable resistance. Can be instantly closed by pressure.

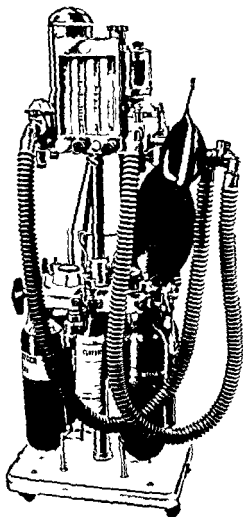


Fig. 23—The Giles Apparatus—portable assembly  
(British Oxygen Co. Ltd.)

- on a spring plunger so is very useful for assisted breathing when a semi-closed circuit is used. Obtainable from Longworth Instrument Co. Ltd. Abingdon on Thames.
- v. THE AIRMED APPARATUS (Fig. 22) —This is an all purpose machine available in both hospital and portable forms. The

## Continuous flow Machines—Boyle's continued

*Expiratory Valves*—These are one way spring loaded valves. They should have minimal resistance to expiration and during spontaneous breathing should always be fully opened as their setting determines the mean pressure in the anaesthetic circuit and in the patient's respiratory tract. Their opening pressure must however be greater than the collapsing pressure of the reservoir bag or the bag will not act as a reservoir. The amount of carbon dioxide rebreathed

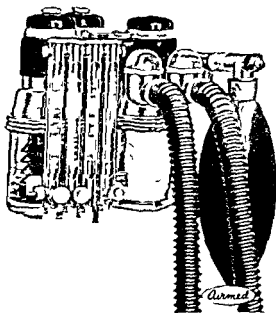


Fig 2.—The Magill nitrous oxide TFC model (Armed Ltd)

depends more on the provision of a plentiful flow of gases to the patient than on the tension of the expiratory valve.\*

An expiratory valve may be combined with a suitable flap making it into a non return or one way valve†

There are four types—

- i The Magill
- ii The Coveter Heidbrink (Fig 3)
- iii The McHesson this can be set to blow off at definite pressures

- iv The Salt valve (Fig 21) Consists of a thin fibre disk on a knife-edge seating Not spring loaded so opens without appreciable resistance Can be instantly closed by pressure

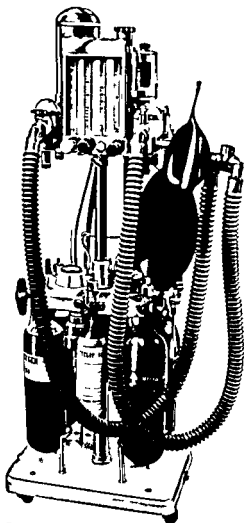


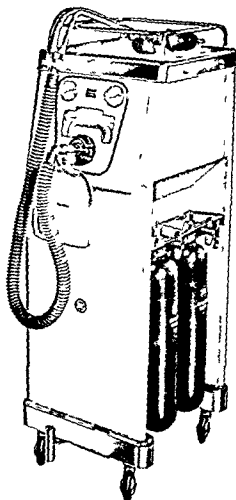
Fig 23—The Gillies Apparatus—portable and military  
(British Oxygen Gas Co. Ltd.)

- on a spring plunger so is very useful for assisted breathing when a semi closed circuit is used Obtainable from Longworth Instrument Co. Ltd. Abingdon on Thames
- b THE AIRMED APPARATUS (Fig 22)—This is an all purpose machine available in both hospital and portable forms The



*Continuous flow Machines—The Airmed Apparatus continued*

makes stress its robust construction minimal resistance to breathing safety in that trilene cannot contaminate soda lime compactness and simplicity It is a compound



*Fig. 4.—The Wallin gas-oxygen apparatus Model No. III  
(British Oxygen Gases Ltd.)*

machine which can be used with a straightforward semi-open circuit or with a closed circuit

c. THE GILLIES MACHINE (Fig. 23)

## 2. INTERMITTENT FLOW MACHINES —

a. THE WALTON MACHINE — Has been made in four models Nos I II III (Fig. 4) and IV (Fig. 5). All are reducing

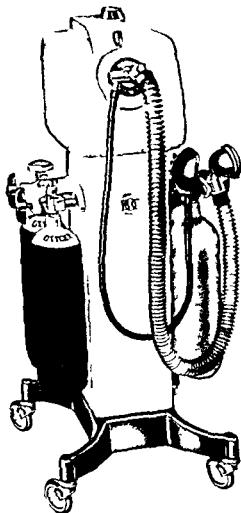


Fig. 25 — The Walton gas-oxygen apparatus, Model I No. IV.  
(British Oxygen Co. Ltd.)

valves lower pressure of issuing gases to 5 to 7 lb. to the square inch. Nitrous oxide and oxygen are led into separate bags which empty with the patient's inspiration, automatically fill again and remain full until emptied. A lever moving

## Intermittent flow Machines—The Walton continued

against graduations controls the percentage of oxygen to be inhaled with the nitrous oxide. A foot lever controls the pressure of the set percentage mixture. A rebreathing bag and vaporizing bottle can be used and a direct oxygen pressure supply is provided. These machines are used in dentistry and can also be employed to produce analgesia in obstetrics.

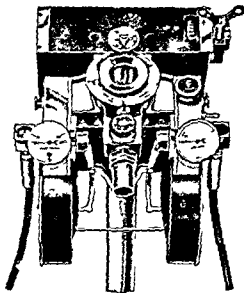


Fig. 6—The head of the McKesson machine (A. Charles & Co. Ltd.)

**THE MCKESSON MACHINE (Fig. 2c)**—Designed and manufactured by Dr. E. I. McKesson, a pioneer anaesthetist of Toledo, Ohio, U.S.A. He was born in 1881 and died in 1935. He classified the signs of anaesthesia with nitrous oxide and oxygen and introduced the method of secondary saturation (q.v.).

Reducing or regulating valves set at 60 lb. to the square inch admit gas and oxygen to two bags enclosed in metal drums at equal pressures. From these bags, which empty only on inspiration, gases pass to a percentage mixing chamber, the proportion being controlled by a dial. Pressure of issuing gas from the mixing chamber varies between 0 and 40 mm. Hg. With pressures above 0, gases are released independently of the patient's inspiration and the machine becomes like a continuous flow apparatus. Other features are a direct oxygen pressure supply which can be used to inflate the chest; a circle type carbon dioxide absorbing circuit; flow meters for carbon dioxide, basal oxygen and cyclopropane; and independent of the closed circuit

a rebreathing bag which may be of the concertina type with control of its volume and pressure. Expiratory valves are placed on the machine and near the mask. There is an ether vaporizing bottle.

To use the machine for ordinary induction turn on the cylinders and see that both nitrous oxide and oxygen register 60 lb to the square inch on the dials on the head of the machine. Turn percentage mixture to 0 per cent oxygen pressure to 3-5 mm Hg and open the expiratory valve near the mask. Gently lower mask to the patient's face until an airtight fit is secured. He breathes in 100 per cent nitrous oxide and oxygen can be added when it is indicated at any desired percentage.

- c THE PORTANÆST—This is a small portable intermittent flow machine looking like a wireless set. It will give variable percentages of the two gases nitrous oxide and oxygen at variable pressures with variable rebreathing. Oxygen can be supplied to the mask under pressure. It is similar to the head of the Walton III machine.

#### Methods of Administration —

- 1 NITROUS OXIDE AND AIR—For this one of the machines described above can be used. More simple is an assembly including a three way stopcock, mask, bag and tubing and a gas cylinder. The three way stopcock is arranged so that inspiration is either from the air (indicator mark AIR) or from the bag (VALVES) while expiration is either into the air (VALVES) or into the bag for rebreathing (NO VALVES).

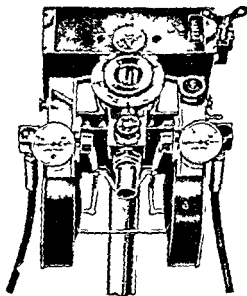
The bag is partially filled with nitrous oxide, some of which can be allowed to flow on to the patient's face as it is  $1\frac{1}{2}$  times heavier than air. Contact is made between mask and face with the lever set at VALVES, thus inspiration is from the bag, expiration into the air. When the pure nitrous oxide produces surgical anaesthesia (onset of regular respiration, abolition of eyelid reflex) the lever is turned to AIR so that a breath of air is taken and is immediately turned back to VALVES. Air is subsequently given every 4-5 breaths as shown by the signs of anaesthesia. If the administration is a long one, gas can be economized by breathing to and fro into the bag with occasional breaths of air. If the mask is removed from the face a period of anaesthesia of 40-60 seconds follows with a shorter subsequent stage of analgesia. If the mask is kept in place anaesthesia can be maintained for five or ten minutes and longer in skilled hands. A nasal inhaler can be substituted for a face mask for intra-oral operations.

Premedication and mechanical restraints such as knee and wrist straps favour a smooth anaesthesia.

- 2 NITROUS OXIDE AND OXYGEN—A smooth anaesthesia with nitrous oxide and oxygen requires a machine capable of delivering accurately known proportions of the gases. A difference of 1 per cent or 2 per cent may make or mar the whole procedure. Premedication must be adequate while sufficient time must be

*Intermittent flow Machines—The Walton continued*

against graduations controls the percentage of oxygen to be inhaled with the nitrous oxide. A foot lever controls the pressure of the set percentage mixture. A rebreathing bag and vaporizing bottle can be used and a direct oxygen pressure supply is provided. These machines are used in dentistry and can also be employed to produce analgesia in obstetrics.



*Fig 5f* —The head of the McHesson machine (A. Charles King Ltd.)

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- c. **THE PORTANÆST**—This is a small portable intermittent flow machine looking like a wireless set. It will give variable percentages of the two gases, nitrous oxide and oxygen, at variable pressures with variable rebreathing. Oxygen can be supplied to the mask under pressure. It is similar to the head of the Walton III machine.

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Premedication and mechanical restraints such as knee and wrist straps favour a smooth anaesthesia.

2. **NITROUS OXIDE AND OXYGEN**—A smooth anaesthesia with nitrous oxide and oxygen requires a machine capable of delivering accurately known proportions of the gases; a difference of 1 per cent or 2 per cent may make or mar the whole procedure. Premedication must be adequate while sufficient time must be

Administration of Nitrous Oxide and Oxygen *continued*

allowed to settle the patient this may vary between 5 and 15 minutes

Commence with pure nitrous oxide at sufficient pressure (3-5 mm Hg) or of sufficient volume (10 litres per minute) so that gas falls on to patient's face from the mask held a couple of inches away. As unconsciousness comes on lower the mask until it makes firm and airtight contact with the face so that the exhaled gas is heard escaping from the expiratory valve. During induction avoid rebreathing as nitrogen should be eliminated as rapidly as possible. See that the expiratory valve is fully open otherwise a feeling of suffocation will result. Quicker deeper breathing indicates mild hypoxia while automatic breathing loss of eyelid reflex and perhaps slight cyanosis indicate surgical anaesthesia. Oxygen is added at first in 5 per cent concentration later in large amounts up to 10 per cent or even 15 per cent. It is often wise to skimp the oxygen during induction and to be adequately free with it during maintenance. The longer the anaesthesia lasts the more oxygen will the patient require. If marked signs of hypoxia appear (twitching of muscles, jactitation etc.) give a breath of 25 per cent or 50 per cent oxygen then carry on with a mixture 1 per cent or 2 per cent richer in oxygen than before. This may need to be repeated more than once. To determine whether patient is hypoxic under nitrous oxide and oxygen give a few breaths of pure oxygen. A previously hypoxic patient will show a temporary fall in blood pressure this is the oxygen depression test and can be employed even if the patient's breathing is controlled\*. Asthenic patients show signs of oxygen lack much earlier than the normal and are usually easier to control demanding a higher oxygen percentage.

Induction is more difficult than maintenance

If in doubt as to depth of anaesthesia (is movement reflex and resistive—too light? or spastic and rigid—too deep?) give a breath or two of oxygen rich mixture. If too light the muscular reaction is made worse if too deep it is improved. A patient with marked hypoxia when given an oxygen rich mixture to breathe will develop a temporary apnoea. This can be used as a test of anaesthetic depth.

If addition of a volatile agent is necessary minimal vaporization of the latter is commenced at the first sign of increased respiratory rate. After a few breaths oxygen is gradually added until it is present in at least 20 per cent concentration. In these circumstances there is no justification for even slight hypoxia. Sufficient supplement is added for the needs of the operation if it is kept minimal with the patient in or above Stage III Plane 1: it is true nitrous-oxide-oxygen anaesthesia with supplement. But if greater depth is reached it is probable that the anaesthesia apart from the induction is due mostly to the

supplement the gases merely acting as a vehicle to carry over the vapour of the volatile agent

In addition to the volatile agents thiopentone makes a most valuable supplementary anaesthetic. It can be given either before or after the nitrous oxide and oxygen. Hypoxia must not occur. Nitrous oxide and oxygen (70 per cent to 30 per cent) with minimal trilete and intermittent thiopentone is a mixture which can be used for a vast range of non-abdominal operations and for these too if a muscle relaxant is used in addition.

Small doses of pethidine (20 mg) can also be used to supplement gas and oxygen. It can be given to patients in the dental chair.

**REBREATHING**—This has two advantages. (a) It economizes gases. (b) It prevents depletion of carbon dioxide from the blood which is encouraged by the increased respiratory rate and depth consequent on hypoxia but it is becoming evident that in all but the shortest anaesthetic procedures rebreathing should be avoided. Rebreathing can be prevented by the use of a non return valve.

Total rebreathing with carbon dioxide absorption is not very satisfactory when nitrous oxide and oxygen are used alone as the addition of the basal oxygen may upset the final percentage of the mixture. With the addition of a little supplementary anaesthetic such as ether the closed circuit is permissible but is probably not as efficient a means of reducing the tension of carbon dioxide inhaled by the patient as a semi-closed circuit with a non return valve.

**RESPIRATORY OBSTRUCTION**—Can be avoided by (a) Careful holding of the lower jaw (b) A pharyngeal airway (c) A nasopharyngeal airway (d) An endotracheal tube.

Airways and tubes should be smeared with lubricant containing a topical analgesic e.g. metycaine 2 per cent amethocaine 1 per cent nupercaine 2–10 per cent xylocaine 2 per cent. Their introduction may cause temporary gagging. A piece of Magill endotracheal tube size 8–10 equal in length to the distance between the nostril and the external auditory canal can be used as a nasopharyngeal tube its distal end lying just above the glottis while its proximal end protrudes from the nostril.

**RESPIRATORY ARREST**—This may be due to —

- 1 Respiratory obstruction and is treated accordingly
- 2 Breath holding in light anaesthesia—treatment usually unnecessary
- 3 Apnoea due to severe hypoxia. Treatment is by inflation of the lungs with oxygen. See that the airway is clear close expiratory valve hold mask firmly on to face and see that oxygen under pressure makes the chest inflate either by touching the direct pressure oxygen button or by compressing the reservoir bag previously emptied and filled with pure oxygen. Breathing pure oxygen raises the oxygen dissolved in blood by 11 per cent. Respiratory arrest usually precedes cardiac arrest so resuscitation by



**Administration of Nitrous Oxide and Oxygen continued**

oxygen inflation is usually successful. Should it not be so other measures must be taken (*see Chapter IV*)

- 4 Apnoea due to a sudden high oxygen tension following a period of hypoxia. It results from a depression of the aortic and carotid body chemoreceptors by the high oxygen tension which removes their reflex stimulating effect on the respiratory centre

**SECONDARY SATURATION**—When anæsthesia is induced with nitrous oxide the oxygen in the circulating blood is rapidly displaced by the gas this is primary saturation. When the oxygen in the tissues is displaced as well it is termed secondary saturation. This is brought about gradually during maintenance of every nitrous-oxide-oxygen anæsthesia of any real duration. It can also be brought about rapidly if speed is essential or if extra muscular relaxation is necessary. It is this rapid saturation which is usually known as secondary saturation. It is a dangerous technique in the tyro's hands and for safety requires a good machine and an experienced anaesthetist. It is now of historical interest only.

**Partial Secondary Saturation**—Give 100 per cent nitrous oxide until severe hypoxia occurs. Then give a breath of 50 per cent nitrous oxide with 50 per cent oxygen which is often followed by a short period of apnoea. Then go over to 100 per cent nitrous oxide and repeat the process several times eventually coming back to 7-10 per cent oxygen in nitrous oxide. If depth cannot now be easily maintained repeat the process.

**Complete Secondary Saturation**—This dramatic technique was originated by McKesson. Give pure nitrous oxide until profound hypoxia with widely dilated pupils, muscular spasms and almost complete respiratory arrest occurs. Now give 100 per cent oxygen so that the lungs are filled with this at the next flagging inspiration. Should inspiration not occur spontaneously and should the full bounding asphyxial pulse change to a small irregular one the lungs are at once inflated with pure oxygen. With the addition of oxygen the pupil at once gets smaller and the muscles relax. After a short apnoea from lack of respiratory drive by the aortic and carotid body chemoreceptors breathing recommences and a mixture of 5-10 per cent oxygen is given in nitrous oxide. If relaxation is still inadequate the whole procedure must be repeated. This technique probably has few justifications to-day. It has given place to a technique utilizing nitrous oxide with at least 25 per cent of oxygen supplemented with thiopentone, pethidine or minimal trileure or ether. Unless the oxygen percentage from the machine is at least 25 per cent alveoli are likely to contain less than 20 per cent of oxygen.

**Carbon Dioxide Elimination in Anæsthetic Circuits**—It is now usually recognized that carbon dioxide

in so far as is possible from the anæsthetic gas mixture inhaled by the patient. The closed circuit with absorption of carbon dioxide by soda lime is not the most efficient way of achieving this and a lower concentration of carbon dioxide is inhaled if a unidirectional circle type absorber is used with soda lime as absorbent in a semiclosed system and with a total gas flow of not less than 7 litres a minute in adults and with the expiratory valve which can be fully opened during expiration as near to the patient's lips as possible\*. The Magill attachment (p. 154) used with a flow rate of at least 7 litres a minute limits carbon dioxide rebreathing to a satisfactory level in the average adult. The conversion of the continuous flow machine for use with a non-rebreathing technique necessitates the incorporation of a one way valve such as that of Ryan (see p. 154). Bullough achieves the same end by using one of the flap valves of the Boyle absorber†. The non-rebreathing technique has the following advantages:

- (1) It prevents inhalation of the expired gases.
- (2) The inhaled mixture is not diluted with water vapour, nitrogen or carbon dioxide and is virtually the same as that delivered from the machine.
- (3) The blood carbon dioxide tension is not raised.
- (4) Nitrogen is rapidly eliminated.
- (5) The respiratory minute volume of the patient can be measured against the total gases issuing from the flow meters of the machine.

The Ayre T piece technique‡ will reduce the inhaled carbon dioxide to reasonable proportions if its internal diameter is 1.25 cm and the capacity of the reservoir tube is 3 ml per inch. The fresh gas inflow must vary between 15 and 6 litres per minute and the capacity of the reservoir tube must vary between 72 and 150 ml—depending on the respiratory minute volume of the patient.

**Nitrous Oxide and Oxygen at High Altitudes**—At high altitudes e.g. over 1 mile it has been suggested§ that the patient should be given at least 30 per cent of oxygen and should inhale an atmosphere containing oxygen at a tension of at least 150 mm Hg.

#### Some Abnormal Types of Patient—

- 1 Patients who are frightened and have a poor command of themselves. These are difficult to control and will need premedication.
- 2 Patients who resist all anæsthetics. Chronic alcoholics, vigorous young men. Supplements are often necessary to control such patients.
- 3 Children under 4. These are not easily managed with nitrous oxide-oxygen because of (a) Emotional reaction (b) High metabolic rate (c) Small volume of circulating blood which causes rapid changes in level of anaesthesia and therefore a small margin between too light and too deep anaesthesia.

Oxygen should be used from the beginning—about 5 per cent later increasing. Premedication most useful. Minimal trilene very helpful as a supplement for short operations.

\* Bracken A. *Proc Roy Soc Med* 1956 49 213

† Bullough J. *Br J Anaesth* 1955 27 181

‡ Ayre P. *Ibid* 1956 28 5

§ Weaver R. H. and Virtue R. W. *Anesthesiology* 1955 16 37

**Administration of Nitrous Oxide and Oxygen continued**

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**Carbon Dioxide Elimination in Anæsthetic Circuits**—It is now usually recognized that carbon dioxide

20 per cent is a suitable basis which may need altering to suit individual patients. Less than 20 per cent of oxygen will if used for any length of time cause foetal hypoxia. Pethidine is a useful drug to combine with gas and oxygen if delivery is not expected within 3-4 hours.

**ANALGESIA DURING SECOND STAGE OF LABOUR**—Four or five breaths are taken commencing just before the onset of the pain. After the last inspiration the breath is held and patient bears down. If the pain is a long one she may get out of breath before its cessation. In these cases three or four quick inspirations are taken followed by bearing down during the remainder of the contraction. Late second stage pains are fairly regular and it is often possible to commence inhalation a minute before the pain is expected and to continue until the pain is maximal followed by bearing down. In this way the blood is saturated with nitrous oxide during the most agonizing phase of the contraction. It must be remembered that when the baby is newly born the oxygen content of the blood from its cord can be greater than that of the maternal venous blood. When the mother receives nitrous oxide and oxygen the baby usually breathes spontaneously on delivery.

Self administration is reasonable in early phase of labour but when the second stage nears its end the anaesthetist should take over.

A little volatile supplement may be necessary for the crowning of the head, trlene being very suitable.

In self administration the patient must be sure to make a leak proof junction between mask and face.

**ANALGESIA DURING THIRD STAGE OF LABOUR**—Manual expulsion of the placenta can be made tolerable with analgesia but perineal and vaginal repair requires anaesthesia.

Intermittent flow machines are very suitable for obstetrical analgesia.

If a continuous flow machine is used gases are economized if some rebreathing is used and if the last expiration is caught in the rebreathing bag and held there until the first inspiration of the succeeding pain.

**Nitrous Oxide-Air**—The Minnitt machine and its modifications are used for this. The patient holding her own mask and using it as described above. The machines are intermittent flow type and are arranged so that they deliver nitrous oxide 45 per cent in 55 per cent air approximately. In some varieties the percentages can be varied. In some others a small air port is kept closed by the patient's finger so that if she becomes unconscious the finger slips and air is breathed instead of the mixture. Care must be taken to instruct the patient in the technique before the onset of labour.

Bad results are usually due to lack of co operation in hysterical patients. Leaks between face and mask, a worn washer between the cylinder and the apparatus, leaking corrugated tubing, a sticking expiratory valve and lack of care and interest on the part of the attendant, starting the administration with each pain too late.

**Some Abnormal Types of Patient continued**

- 4 Patients who are anæmic They do not tolerate hypoxia Induction should be slow with a high percentage of oxygen 25-30 per cent gradually increased or decreased according to the patient's needs Cyanosis may be a grave sign With a hæmoglobin of 30 per cent or less death may occur before the onset of cyanosis
- 5 Patients with decompensated heart disease These should be treated like the anæmic group It is often a good plan to precede anaesthesia by the inhalation for ten minutes of 100 per cent oxygen
- 6 Patients with hypertension With good oxygenation these patients do well Struggling during induction should be avoided
- 7 Patients in shock They are usually easily controlled but tolerate oxygen lack badly
- 8 It is seldom desirable to anaesthetize patients who are physically handicapped by nitrous-oxide-oxygen unsupplemented except for short minor operations

**Advantages of Nitrous Oxide —**

- 1 Rapid induction and recovery
- 2 Relative absence of post-operative nausea etc
- 3 Absence of toxic effects and relative safety if hypoxia is avoided
- 4 Safety in absence of gross hypoxia
- 5 Not explosive
- 6 Not unpleasant to inhale

**Disadvantages of Nitrous Oxide —**

- 1 Its weak anaesthetic properties
- 2 Need for heavy apparatus
- 3 Expense

Straight nitrous oxide-oxygen anaesthesia is dwindling in popularity Supplements such as thiopentone trilene and pethidine are used more and more frequently This last drug given intravenously in doses of 25-100 mg shortly before induction of anaesthesia is often most helpful before nitrous oxide and oxygen

**NITROUS OXIDE IN OBSTETRICS**

**Nitrous Oxide-Oxygen —** Nitrous oxide does not interfere with uterine contractions nor has it any effect on the fœtus The Russian Khkovich introduced nitrous oxide into obstetrics in 1880 Guedel devised the first machine for self administration of nitrous oxide in obstetrics in 1910

**ANALGESIA DURING FIRST STAGE OF LABOUR —** In the early first stage if patient complains of pain it is better to give a sedative such as tinct opii 14 minims or chloral hydrate 20 gr It is nevertheless better to start self administration of gas too early rather than too late Before gas is given see that the machine is working well that cylinders contain gas and that dentures are removed Inhalation must begin some seconds before the onset of the pain If the patient holds her own mask it will fall from her hand should unconsciousness supervene this is a safety factor Nitrous oxide 80 per oxygen

patients must be certified by the doctor as fit to receive analgesia and a third person agreeable to the patient must be present during the labour. Midwives are also allowed to use trilene from certain approved inhalers e.g. The Emotril the Tecota Mk 6.

The Portanæst machine is a useful machine for analgesia in labour but cannot be used by a midwife working alone.

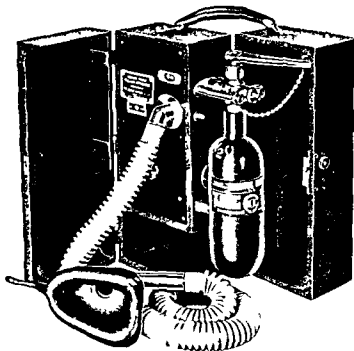


Fig. 28.—The Minnitt Minor apparatus (British Oxygen Gases Ltd.)

Vapour of volatile anaesthetics can be added to the gas and air for resistant patients or to produce anaesthesia during delivery; the machines alone are only designed to produce analgesia.

**THE ORIGINAL MINNITT APPARATUS (Fig. 27)**—This was a modification of the McKesson oxygen therapy apparatus and was introduced in 1933. A reducing valve steps down the gas pressure to 60 lb to the square inch and gas is led from it into a rubber bag enclosed in the familiar McKesson drum. After leaving the bag gas is mixed with about 50 per cent of air. An expiratory valve is provided and the machine is of the usual intermittent flow type, the patient's inspiration controlling the flow of gas. The weight without cylinder is 15 lb, while the weight of a 100 gallon cylinder is 8.9 lb empty, 10½ lb full.

**Nitrous Oxide-Air in Obstetrics continued**

All pupil midwives must attend lectures on gas-air administration during labour and are asked questions on the subject in their exams. Midwives when working without medical supervision

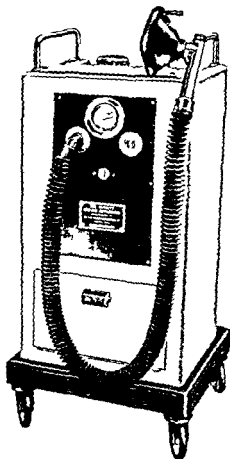


FIG. 27.—The Minnitt apparatus for gas-air anaesthesia in obstetrics.  
(British Oxygen Co. Ltd.)

are only allowed to use certain gas-air machines including the Minnitt (Fig. 27), Minnitt-Walton, Minnitt-Minor (Fig. 28), Talley (weight 8 lb.), Jecta (weight 17 lb.) and Amwell. Each gives 50 per cent gas in air. They are not allowed to use C.M. attachments or trilete vaporizers attached to gas-air machines. Their

Fœtal distress should contra indicate the continuance of gas-air analgesia. It is shown by —

- 1 A fœtal heart rate becoming progressively slower or faster between pains
- 2 When fœtal heart does not increase after contractions as previously it did



Fig 30—Tject gas air machine (Medical and Industrial Equipment Ltd)

- 3 When fœtal heart is irregular between pains

These gas-air machines are also useful for changing painful dressings and to produce analgesia during dental drilling

### NITROUS OXIDE IN DENTISTRY

Major dental operations are best done in an operating theatre on in patients

For operations in the dentist's chair premedication may be difficult as the patient is ambulatory. A hypnotic the night before operation (medinal 5-10 gr) together with aspirin (10-15 gr) before the extraction is a good plan. Seconal allonal and nembutal are used by some. The stomach and bladder should be empty, the nose should be blown and dentures should be removed. A stethoscope should be applied to the patient's chest to reassure him. Heavy footwear should be removed and the patient should sit upright in the chair.



*Nitrous Oxide-Air in Obstetrics continued*

**THE MINNITT WALTON APPARATUS**—An Adams valve reduces gas pressure to 4–5 lb to the square inch. The rubber bag is enclosed in a metal box. The working principles are the same as in the original Minnitt machine.

The C M attachment is an apparatus capable of delivering two breaths of pure nitrous oxide from a bag holding 2 litres of the gas (Fig. 29). Time taken to refill the bag is about one minute.

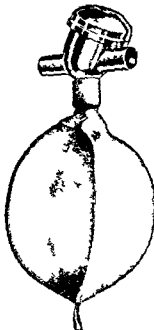


Fig. 29.—The C M attachment of the Minnitt apparatus.  
(British Oxygen Co. Ltd.)

thus overdosage of gas is avoided. Following the breaths of pure gas the patient continues to breathe gas and air from the machine together with pure gas 1 litre/min.

Elam's Wellhouse machine delivers pure nitrous oxide initially but automatically switches to gas-air later.

**THE JECTA GAS-AIR MACHINE**—This is fitted with a regulator which reduces the pressure of nitrous oxide to 60 lb to the square inch and utilizes the jet and Venturi system to obtain the desired mixture. An intermittent mechanism is provided while a safety device shuts off the gas supply should the air orifice become occluded. A nitrous oxide and air mixture passes to the patient via a non return valve breathing tube and face piece (Fig. 30).

the nose piece and the face is aimed at and the establishment of nasal breathing is indicated by the sound of gas issuing from the expiratory valve. When breathing quickens a breath of air can be given either by opening the air port or by slightly lifting the nose piece from the face. It is however wrong to give much air until the onset of surgical anaesthesia. Because the blood takes about 10 seconds to travel from the lungs to the tissues air should be given 3 or 4 breaths before its need becomes apparent. This requires judgement. With the onset of regular respiration and the disappearance of the eyelid reflex surgical anaesthesia is shown to be present (*see SIGNS OF NITROUS OXIDE ANAESTHESIA* pp. 144 et seq.)

The mouth is now packed with either a marine sponge or a strip of gamgee tissue about 6 in.  $\times$  3 in. care being taken to avoid pushing the tongue back to the posterior pharyngeal wall and so obstructing breathing. The pack helps to prevent debris from being inhaled; it absorbs blood and mucus and it hinders mouth breathing. To maintain a free airway the jaw may have to be pushed forwards especially if the dentist is working on lower molar teeth. Air is given every few breaths according to the patient's needs which depend on the signs of nitrous oxide anaesthesia. Extraction must not commence until depth is adequate and nasal respiration is established. Avoid giving too much air during induction and too little during maintenance.

During recovery the patient should not be fussed over but should be quietly left to regain consciousness.

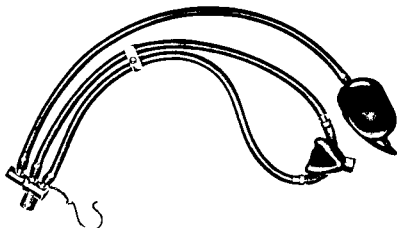
**DIFFICULTIES**—The chief of these are (a) Inability to establish nasal breathing (b) Inability to maintain nasal breathing (c) Patients with respiratory obstruction due to infection in the floor of the mouth Ludwig's angina etc.

- a *Inability to establish Nasal Breathing*—May be due to nervousness nasal obstruction or plain stupidity (of the patient!)
    - 1 Apply mouth inhaler which delivers gas in addition to nasal inhaler. The mouth should not be blocked with a hand towel or obturator as a feeling of suffocation is thus produced.
    - 2 Induce with a face-piece. Once surgical anaesthesia is established breathing becomes nasal and maintenance can be carried on with the nose piece.
    - 3 Deliver the gas under pressure by increasing the gas flow and squeezing the bag during inspiration at the same time tightening up the expiratory valve.
  - b *Inability to maintain Nasal Breathing*—This may be due to surgical stimuli applied at too light a plane of anaesthesia. It may be due to fear. The condition easily wrecks the anaesthesia as the patient getting no nitrous oxide becomes lighter. The dentist must be asked to pause while anaesthesia is deepened. A nasopharyngeal tube may be helpful.
- The Patients who resist Anaesthesia*—These may include (a) The frightened (b) The tough and sturdy (c) The alcoholic

*Nitrous Oxide in Dentistry continued*

**Nitrous Oxide-Air**—A dental prop is inserted and the chair so arranged that the patient's head is in line with his body. The whole chair can be tilted backwards if desired. If the foot plate is removed the patient cannot push against it. If difficulty is expected a strap passed loosely over the pelvis can be tightened when the patient becomes unconscious.

For one or two easy extractions a face piece can be employed but for all others a nasal inhaler such as Trewby's the British Oxygen Company's (*Fig. 31*) Harn's or McKesson's should be



*Fig. 31*—Oro-nasal inhaler (British Oxygen Gases Ltd.)

used. In addition to the nasal inhaler a breathing bag and tubing and a nitrous oxide cylinder are necessary. There is no need to have a reducing valve.

The administration of nasal gas is not easy—it requires study and experience.

**POSITION OF ANÆSTHETIST**—He should stand on the left side and a little behind the patient whose head should be supported while the nasal inhaler is held securely in place to prevent leaks. The lower jaw should be held forwards and counter pressure should be exerted on the vertex when the upper teeth are being extracted. The patient's head should be turned to the opposite side when the lower teeth are receiving attention.

**TECHNIQUE**—The patient is told to breathe easily through the nose. This is most important and is worth spending some time over. He should close his eyes. A moderate flow of gas is turned on (8-15 litres a minute) and the nose piece gradually lowered after being held an inch or two away during induction of analgesia. The nose piece must not be pushed upwards lest the nostrils become occluded. An airtight junction between

the nose piece and the face is aimed at and the establishment of nasal breathing is indicated by the sound of gas issuing from the expiratory valve. When breathing quickens a breath of air can be given either by opening the air port or by slightly lifting the nose piece from the face. It is however wrong to give much air until the onset of surgical anaesthesia. Because the blood takes about 10 seconds to travel from the lungs to the tissues air should be given 3 or 4 breaths before its need becomes apparent. This requires judgement. With the onset of regular respiration and the disappearance of the eyelid reflex surgical anaesthesia is shown to be present (*see SIGNS OF NITROUS OXIDE ANAESTHESIA pp 144 et seq*)

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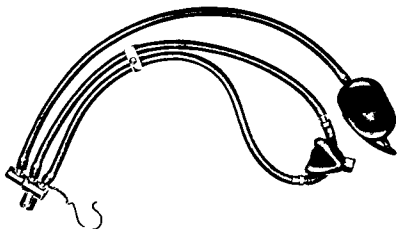


Fig. 3 —Oro-nasal inhaler (British Oxygen Gases Ltd.)

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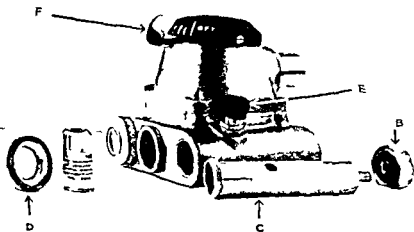
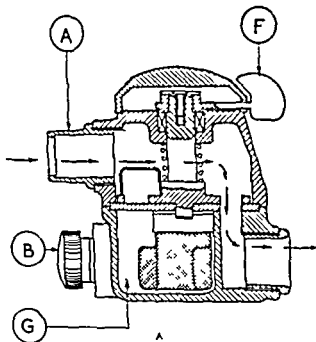


Fig. 32—A, Section of the Finn's Trilene Vaporizer. B, The Finn's Trilene Vaporizer (B & J Anesthesia).

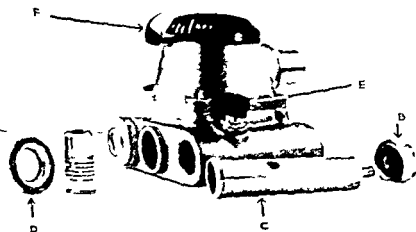
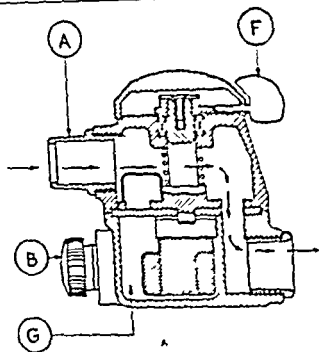
**Nitrous-Oxide—Air in Dentistry—Technique continued**

There may be difficulty in nasal breathing during induction or profound hypoxia may be required to subdue them. Premedication is of great value and nembutal 1½ gr given to a nervous adult will often work wonders. Children can be similarly medicated giving ½ gr of nembutal per stone of body weight. One of the following methods may be employed to deal with such resistant patients —

- 1 If trouble is foreseen anaesthesia can be quickly and pleasantly induced with 0.25–0.5 gr of thiopentone or 0.4–0.6 g of buthalitone intravenously. Injection is rapid with the patient sitting in the chair the strap being fastened to prevent slumping. Maintenance of anaesthesia is by nitrous oxide and air. With correct dosage patients recover in a matter of minutes.
- 2 A 3 ml ampoule of vinesthene can be broken on to a towel placed over the patient's mouth. This helps induction while the usual gas is given for maintenance.
- 3 Ethyl chloride can be used in place of vinesthene using 3–5 ml.
- 4 An ether-soaked sponge can be placed in the patient's mouth after induction with gas. Nasal gas is proceeded with. Sturdy patients are very little upset by the ether.
- 5 Trilene can be used. A Rowbotham's bottle can be attached to the exit tube of the gas machine or a Finnie vaporizer employed. This utilizes ampoules of trilene. (See Fig. 3.) A vapour concentration of 0.05 per cent to 0.1 per cent is adequate in most cases with nitrous oxide and oxygen. A little goes a long way—overdosage must be avoided.
- 6 Pethidine 25 mg can be given intravenously before induction. This quiets the patient and does not cause much hangover.

**Nitrous Oxide—Oxygen**—A gas machine is often preferred for dental work and usually will enable a smoother anaesthetic to be given than when gas and air is used. Induction is with nitrous oxide as in the nitrous-oxide-air technique. When surgical anaesthesia is reached 6–10 per cent of oxygen is added. Here again oxygen is kept low during induction and given more liberally as the patient becomes settled. The nasal inhaler is used with its expiratory valve open. If anaesthesia is too light a breath or two of pure nitrous oxide is given followed by a mixture 1 per cent less rich in oxygen. Similarly with an anaesthesia too profound a breath of oxygen rich mixture is followed by 1 per cent or 2 per cent more oxygen. A difference of 1 per cent or 2 per cent may make a vast difference to a smooth anaesthesia.

In anaesthetic resistant patients volatile supplements can be added in small quantities from the vaporizing bottles. Trilene is



A. Section of the Flowmeter valve. B. The Flowmeter valve (Bore 1 inch).



**Nitrous Oxide—Oxygen in Dentistry** *continued*

especially useful for this work. Bourne has reported the use of cyclopropane in dentistry and has designed a portable apparatus employing minute sparklet cylinders for its administration (see Chapter VII)

**Amalgamia** \*—This is a plane between analgesia and anæsthesia and it allows 15–20 per cent of oxygen to be given (apart from a lower percentage for a few seconds during induction). In this plane operation can be performed without pain or the memory of it. When used for children pure gas is given for half a dozen breaths and this is followed by an 85–15 nitrous-oxide-oxygen mixture. Forty or fifty breaths are required before the dental surgeon should be allowed to make his extractions and at this time the breathing is free and regular and the eyelids are relaxed over expressionless eyes. In adults up to 20 per cent of oxygen can be used. Cyanosis, stertor and jactitation are of course unknown and the duration of analgesia can be considerable. Reflex response to trauma may occur but is not accompanied by the sensation of pain or the memory of it.

**Fainting in the Dental Chair** †—Recently attention has been drawn to the possibility of fainting during the administration of nitrous oxide in the dental chair. Should the patient suddenly become pale he must be immediately tilted into the horizontal position. If this is neglected delayed recovery from anæsthesia, permanent cerebral damage from hypoxia or even death may result.

**Analgesia in Dentistry**—This is indicated when painful fillings are to be performed. The patient must co-operate with the anæsthetist so that hysterical or unintelligent patients are unsuited for this type of pain relief.

The nose piece is gently applied and the mouth is kept open. Induction is either by a few breaths of pure nitrous oxide or by a mixture of gas and oxygen. After a few breaths the oxygen percentage is set to about 35. The patient is told to breathe through the nose but to take a breath or two of air through the mouth if he feels himself going too deep or losing consciousness. If pain is felt the percentage of oxygen should be reduced. By asking an occasional question and noting the manner of the reply the anæsthetist is able to maintain an even plane of analgesia. Should second stage anæsthesia develop the patient may become uncontrolled.

The subjective feelings are of euphoria, great awareness of sounds and relief from pain.

The technique is useful for changing painful dressings, etc.

**TRILENE** and air is being developed as an analgesic in both dentistry and obstetrics. The Williams-Hill Trilene Inhaler for self administration is useful. Overdose is impossible since delivery of vapour is dependent on the muscular action of bulb squeezing. Trilene cannot be blown into the delivery tube and overfilling is impossible.

The Trilite Inhaler can also be used to produce analgesia for conservative dentistry

**VENON**—This is one of the rare gases of the atmosphere and it was used in anaesthesia first of all by Cullen and Gross in 1951\*. It appears to have a potency similar to that of ethylene and causes neither respiratory nor cardiovascular depression. It should be given with oxygen in a 20-80 per cent mixture and is non flammable†

## CHAPTER IX

# ACCIDENTS OF INHALATION ANÆSTHESIA AND HOW TO TREAT THEM

- Vomiting**—Vomiting is a complex reflex act controlled by a centre in the medulla near the vagal nucleus and the respiratory centre. Afferent impulses reach it from any part of the alimentary canal, the heart, inner ear or brain. At the beginning of the act of vomiting, while the pylorus shuts, the stomach relaxes and is compressed against the posterior abdominal wall and the other abdominal viscera by the descent of diaphragm and the contraction of the anterior abdominal wall muscles. The cardia now opens and stomach contents are forced into the oesophagus, the mouth and sometimes even the nose. While the glottis goes into spasm during the expulsive phase, it soon relaxes so that aspiration of stomach contents into the bronchial tree is almost bound to happen in the unconscious patient. Predisposing factors during anaesthesia include (a) Hypoxia (b) Central stimulation during second stage general anaesthesia—either during induction or recovery (c) Irritation of the base of the tongue or pharynx by airways etc.

Regurgitation being a passive act may be silent and unheralded and so even more potentially dangerous than vomiting. Predisposing factors include (a) The head down position (b) A stomach full of fluid. O'Mullane showed‡ that the cardiac sphincter is not affected by relaxants, local analgesia, general anaesthesia or ganglioplegics. The presence of a stomach tube makes the sphincter inefficient while an obstruction in the airway which causes a negative pressure in the pharynx and oesophagus encourages the sphincter to open. It seems that the sphincter acts as a valve rather than as a true sphincter, allowing fluids to pass distally but not proximally (in the absence of a raised intra-gastric pressure). The cricopharyngeal sphincter on the other hand acts as a sphincter normally but as a valve when paralysed.

Cullen, S. C. and Gross, E. G. *Fed. Proc.* 1951 10 290

\* Fink, B. R. *Anesthesiology* 1955 18 29

‡ O'Mullane, E. J. *Lancet* 1954 1 1209

*Vomiting continued*

It gives exit to fluids from the œsophagus to the pharynx, but not in the reverse direction. When paralysed by relaxants it tends to obstruct the passage of fluids from the pharynx to the œsophagus but not in the reverse direction.

When considering this question all anæsthetists should consult the first-class paper by H J V Morton and W D Wylie\* dealing with vomiting during anæsthesia and its relationship to deaths on the table in the causation of which it is one of the major factors.

**CAUSES—**

- a* Vomitable material in the stomach or œsophagus—
    - 1 Inadequate pre-operative preparation of the patient
    - 2 Blood in the stomach following bleeding from ulcer tonsil beds œsophageal varices or during gastric operations
    - 3 Glucose solution mistakenly given to diabetic patients by mouth instead of intravenously
    - 4 In cases of œsophageal disease such as pouch or obstruction
  - b* Vomitable material returned into stomach from bowel as in cases of intestinal obstruction
  - c* When stomach emptying time is delayed—
    - 1 In women in labour
    - 2 In cases of head injury
    - 3 When there is emotional strain associated with pain accident and the incident of hospitalization
    - 4 In seriously ill patients
- The stomach may not be empty eight to twelve hours after the last meal

**DANGERS OF VOMITING DURING ANÆSTHESIA—**These are—

- a* The inhalation of stomach contents into the lungs with sequelæ such as pneumonitis bronchopneumonia atelectasis and lung abscess
- b* Hypoxia due to laryngeal spasm and obstruction of the air passages
- c* Reflex cardiac inhibition from reflexes originating in the bronchi due to acid contamination

**PREVENTION—**Except in the gravest emergency no general anæsthetic should be given to a patient whose stomach may contain vomitable material i.e. a patient who has not been properly prepared for anæsthesia by preliminary avoidance of food and drink. This should be an infallible rule.

When an anæsthetic must be given for urgent reasons a Ryle's tube e.g. size 6 gauge should be passed from the nose into the stomach so that aspiration can take place and if necessary lavage. Information is thus obtained as to the amount and type of material still in the stomach. If the anæsthetist thinks that he can empty the viscus by the tube well and good but in all cases of doubt an œsophageal tube such as size 12 should be inserted into the cocaineized nostril and

thence into the stomach where it should remain until the patient gets his cough reflexes back at the end of the operation. This tube will allow fairly efficient drainage of liquid and semi solid material and so should prevent regurgitation of liquid or semi solid material even if actual vomiting is not prevented.

The insertion of a cuffed endotracheal tube into the larynx before the onset of vomiting or regurgitation is the only safe procedure when dealing with a patient who may have vomitable material in the stomach or oesophagus. In reasonably skilled hands this can be done while the patient is still conscious under topical analgesia. It can also be done if an intravenous thiobarbiturate together with a rapidly acting relaxant is injected with a head up tilt of the patient always provided that the anaesthetist is an experienced worker. Another method is to substitute a 50-50 cyclopropane-oxygen mixture for the intravenous barbiturate.

Induction with a foot-down tilt will make regurgitation less likely to steal up on the anaesthetist but an ill patient will not tolerate this position well. A slow smooth induction using gas-oxygen and a volatile agent helped perhaps with a little carbon dioxide may be safer than a quick induction using thiopentone and a relaxant the head being tilted downwards. A cuffed tube should always be used when in doubt about the contents of the stomach and before induction commences the availability of an efficient suction apparatus source of oxygen laryngoscope endotracheal tubes airways etc must be checked. It is wise to induce these patients on the operating table so that tilting can be employed easily.

An efficient but not a particularly humane method of reducing the risk of aspiration of stomach contents before induction of anaesthesia is to empty the patient's stomach by stimulating vomiting. Apomorphine  $\frac{1}{4}$  gr is dissolved in 10 ml of saline and 1 ml is injected intravenously at short intervals until the patient vomits. Once the stomach is empty nausea is abolished and relatively safe induction of anaesthesia should follow.

**TREATMENT**—*If vomiting occurs* The air passages must if possible be spared contamination by tilting the head downwards or turning the patient on one side. Suction must be used and oxygen carried to the alveoli in the most efficient way that is possible under the circumstances. Endotracheal intubation should only be attempted if relaxation is present and the manoeuvre likely to be quick and easy otherwise hypoxia may be made worse rather than better. Should the airways be contaminated then suction and gravity should be employed to lessen the extent of the insult to the bronchi. Endotracheal suction may be sufficient in mild cases whereas in others suction through a bronchoscope will be required. It may be combined with lavage 10 ml of saline being injected

**Vomiting—Treatment continued**

and aspirated via the bronchoscope and repeated several times. This should not be done immediately but after the patient has recovered from the immediate effects of the catastrophe.

To lessen the dangers of vomiting during recovery the patient should whenever possible be returned to the ward in the so called tonsillar position lying on his side with the bottom arm behind him to prevent his turning on to his back and with pillows under the chest to prevent his rolling into the prone position.

- 2 Coughing**—Occurs most commonly when ether is used and when patient has chemical e.g. due to heavy smoking or infective inflammation of the upper air passages.

Concentration of gases and vapour must be increased slowly. A few breaths of trilene aid in the introduction of ether.

Coughing may also cause trouble during anæsthesia with thiopentone this is best controlled by nitrous oxide-oxygen and minimal trilene. It may be due to irritation of the larynx from regurgitated gastric material or from saliva.

- 3 Excessive Mucus**—This can often be avoided by adequate premedication together with smooth induction.

It should be treated by clearing out the mucus with swabs or a sucker and rotating head to one side.

Stimulation of the pharyngeal and laryngeal reflexes as by artificial airways may produce excessive mucus especially in babies and children. If the table is lowered head down mucus tends to drain away from the hypopharynx. Patients who are moist should be thoroughly sucked out at the end of the operation.

- 4 Respiratory Arrest**—Due to obstruction of the airway or to central respiratory depression.

**TREATMENT**—After establishing the airway using an endotracheal tube if necessary oxygen must be carried to the alveoli—

- 1 By blowing air into the lungs from a face piece
- 2 By forcing oxygen into lungs from a reservoir bag of a gas machine with manual pressure after closure of the expiratory valve
- 3 By use of direct oxygen button on a gas machine or the Stephenson Minuteman
- 4 By manual compression of the thorax
- 5 By grasping the costal cartilages in the epigastric region and expanding the thoracic cage (Viswanathan)
- 6 By Silvester's method of artificial respiration (1858) whereby inspiration is produced by raising the arms above the patient's head with the production of expansion of the thorax and expiration is aided by compressing the arms against the chest
- 7 By Schafer's method of artificial respiration (1903) with the patient prone on the floor and the anaesthetist applying pressure to lower ribs. This is seldom of use in anæsthesia except in dental cases
- 8 By Eve's rocking method (1932)
- 9 By Drinker's mechanical respirator (1909)

- 10 By Holger Nielsen's method of artificial respiration which although unsuitable for use on the operating table should be known to anaesthetists. The patient lies prone with arms overhead elbows flexed one hand on the other the cheek resting on the uppermost hand. The operator kneels on one knee at the patient's head facing his feet. The operator now grasps the patient's arms just above the elbows lifts them and rocks the thorax backwards this allows inspiration to take place. The arms are then dropped the operator placing his hands just below the scapulae air is expressed from the thorax. The cycle is repeated about twelve times each minute allowing no pause between inspiratory and expiratory manœuvres. This arm lift back pressure technique produces a tidal exchange more than double that produced by Schafer's method and blood-oxygen levels should approach 90 per cent of saturation. It can in addition be carried out by persons of frail physique.

Should it be impossible to establish an airway by a pharyngeal or endotracheal tube tracheotomy or laryngotomy may be necessary.

*Laryngotomy*—Easier to perform than tracheotomy but may be followed by permanent narrowing of larynx. A transverse incision of the skin is made between the thyroid and cricoid cartilages with the patient's neck extended. With an introducer or artery forceps the cricothyroid membrane is pierced and the short flat laryngotomy tube introduced and secured. Should not be done in children.

*Tracheotomy*—With the neck steadied in the extended position an incision is made in the midline below the cricoid cartilage. A vertical incision is made in the trachea and the edges are kept open while a tracheotomy tube and introducer are inserted. The inner tube is then placed in position and secured. bleeding points are dealt with after restoration of the airway.

Mediastinal emphysema and pneumothorax are frequent complications of tracheotomy and can be prevented more easily when the operation is planned than when it is done in an emergency. Its cause is the abnormally low intrathoracic pressure resulting from the obstruction and this causes air to be sucked into the wound before the trachea is opened. To avoid this complication an endotracheal tube should be passed before the wound is made this will raise the negative intrathoracic pressure to normal. The tissues and fascial planes during operation should be opened up as little as possible and once the pretracheal fascia is incised the trachea must be opened rapidly so that there will be no time for air to be sucked into the mediastinum. Following operation the airway must be kept patent.

A third measure may be adopted if the patient is *in extremis*. A large bore needle is inserted through the cricothyroid membrane into the larynx and through it either air is rapidly

Respiratory Arrest—Treatment *continued*

injected with a syringe or oxygen is delivered to the needle under slight pressure allowance must of course be made for emptying the lungs

- 5 Atelectasis**—This may occur fairly suddenly during or immediately after the anæsthesia. Its signs are progressive hypoxia and absence of breath sounds over part of the lung area. The diaphragm rises to occupy the space vacated by the lung. It may be caused by reflex bronchiolar spasm which by cutting off the return of blood by the veins could result in œdema of the mucosa and bronchial obstruction. Clinically it is shown by increasing difficulty in breathing or in inflation of the lungs. If the chest is open the infiltration of the lung hilum with a local analgesic solution may do good. Spontaneous atelectasis of the left lung can occur if the right bronchus is accidentally intubated.

Closed pneumothorax has been reported and is presumably due to

- (1) The rupture of lung tissue—the result of excessive coughing or of intermittent positive pressure respiration carried out too vigorously. The treatment is to aspirate air from the pleural cavity. (2) Operations on the neck associated with respiratory obstruction which causes an increase in the negative pressure in the mediastinum during inspiration and consequent trapping of air. This trapped air may rupture into the pleura or spread into the soft tissues of the neck, axilla, face or thoracic wall or along aorta and œsophagus into the abdomen. It may have to be evacuated by blunt dissection into the mediastinum from an incision just above the manubrium sterni.

Treatment consists in inflation of the lungs and if necessary bronchoscopic examination and suction of the upper air passages.

- 6 Surgical Emphysema during Anæsthesia**—This was first reported in 1912 (Woolsey) during insufflation endotracheal anæsthesia. Surgical emphysema commences as a pulmonary interstitial emphysema due to overdistension of the alveoli, i.e. at a pressure greater than 20 mm Hg. The gas tracks along the sheaths of the vessels to the hilum—mediastinal emphysema from which it may spread (a) to the neck, (b) to the abdomen, (c) behind the peritoneum, (d) into the pleura (tension pneumothorax). Once the mediastinal pleura is ruptured very little extra pressure will force gases into the pleural cavity. Tension pneumothorax should always be looked for if surgical emphysema appears. Should circulatory or respiratory difficulty arise in the presence of mediastinal emphysema an incision should be made at the root of the neck anteriorly and gas should be let out by blunt dissection.\*

- 7 Convulsions**—Several types of abnormal muscular action may occur during anæsthesia—

- 1 Deep ether convulsions (*see p 96*)
- 2 Ether clonus—usually occurring in light anæsthesia and disappearing when anæsthesia is deepened. Commonly seen in

the legs and may be stopped by raising thighs leaving legs unsupported

- 3 Epilepsy Intubation may be required to ensure oxygenation
  - 4 Convulsions due to hypoxia e.g. during nitrous oxide anaesthesia the so-called jactitations
  - 5 Convulsions due to local analgesic drugs e.g. lignocaine procaine amethocaine Treat with intravenous thiopentone and oxygen inhalations
  - 6 Tremor associated with the intravenous injection of thiopentone usually a pronator spasm of the arm receiving the injection Addition of more thiopentone together with nitrous oxide and oxygen will usually cure the condition which is commoner after 5 per cent solution than 2½ per cent solution
- 8 Damage to Eyes**—May follow accidental instillation into conjunctival sac of ether blood antiseptic lotion etc splashed from the operation site
- May be due to trauma from tightly applied face mask or friction from gauze or fingers
- The eyes should be closed oiled and protected suitably especially where there is exophthalmos
- Pressure on the eyeballs e.g. from a mask prolonged hypotension and steep Trendelenburg position have all been blamed for causing occlusion of the central artery of the retina
- 9 Status Lymphaticus**—Paltauf (1889) was first to describe the syndrome in recent times but it has been discovered forgotten rediscovered and declared to be non-existent from 1614 onwards A condition occurring in children of fat lymphoid type Alan Moncrieff states that the condition may be suspected if there is a history of attacks of fainting with shortness of breath attacks of head retraction and stridor X-ray evidence of an enlarged thymus gland
- Pathologically there may be enlargement of the thymus hypertrophy of lymphoid tissue and fatty degeneration of the heart and possibly suprarenal cortical deficiency
- Such children may be very sensitive to anaesthetic agents requiring amounts much less than normal Macintosh advises that children who go under without any signs of resistance and who require only small amounts of anaesthetic should be treated with extra special care lest sudden death result from overdosage Ether is probably the safest anaesthetic for such cases
- Many pathologists consider that no such condition exists as a definite entity (Report of British Status Lymphaticus Commission 1931) but it is frequently stated that sudden death during anaesthesia in children is due to status lymphaticus whereas it may well be due to error of technique or judgement on the part of the anaesthetist
- 10 Acute Hypoxia**—This may be associated with cardiac arrest or may by good fortune stop short of this calamity It is most likely to be due to inattention on the part of the anaesthetist Symptoms after anaesthesia may be coma stertorous breathing



*Acute Hypoxia continued*

hyperpyrexia restlessness choreo athetosis and convulsions  
For treatment *see under* CIRCULATORY COLLAPSE (p 183)

**11 Circulatory Collapse**—This when it occurs during anæsthesia may be due to (a) Primary cardiac failure or (b) Secondary or gradual cardiac failure Shock hæmorrhage or air embolus may also cause circulatory failure while pulmonary or cardiac infarction and massive collapse of the lung must be borne in mind

**a PRIMARY CARDIAC FAILURE**—This may take the form of—

1 Ventricular fibrillation consequent on an irritable state of the automatic conductive tissue of the heart This was first described by Ludwig and Hoffa in 1847 and is sometimes known as *delirium cordis* There is complete incoordination of the ventricular muscular fibres and no effective circulation Electrical impulses can be recorded on the electrocardiograph several minutes after cardiac action has ceased

2 Cardiac standstill or asystole

**CAUSES**—

- 1 Reflex mechanisms usually involving vagal afferents and either vagal or sympathetic efferents (vagovagal or vago-sympathetic) These usually occur during light anæsthesia as following endotracheal intubation or too strong an anæsthetic vapour aspiration of gastric acid contents into the bronchial tree pulling on the mesentery etc These reflexes are also potentiated by hypoxia There is cardiac standstill from vagal inhibition and from sympathetic overactivity Short of cardiac standstill the following forms of arrhythmia may be caused (α) sinus bradycardia (β) auriculoventricular nodal rhythm (γ) heart block A proper dose of atropine has a good protective action against these reflexes which are more likely to arise when vagal tone is increased in peptic ulceration jaundice and simple vagotonia with sinus bradycardia
- ii Overdosage or sensitivity to anæsthetic or other drugs e.g. ethyl chloride chloroform or cyclopropane adrenaline toxic doses of digitalis procaine amide neostigmine diiodone hyperkæmia
- iii Mechanical interference with the heart's action in thoracic surgery—cardiac catheterization etc
- iv Hypoxia and hypercapnia both of which may occur without obvious cyanosis It is the duty of the anæsthetist to maintain at all times in so far as is possible the  $pO_2$  and  $pCO_2$  of the blood at normal levels by efficient pulmonary ventilation
- v Cardiac pathology e.g. coronary disease aortic stenosis A V heart block
- vi Air embolus Air can gain access to the venous circulation through any vein if the physical factors are right Thus a neck vein with the head raised or a pelvic vein in the steep Trendelenburg position may if injured cause air bubbles to be sucked in These prevent proper emptying of the right

heart and occlusion of the pulmonary artery. On auscultation a loud pathognomonic murmur is heard with the stethoscope over the heart. Treatment consists in lowering the head and turning the patient on to his left side and aspiration of air from the right ventricle via a long needle inserted either in the fifth interspace to the right or the fourth interspace to the left of the sternum—in each case directed medially.

#### vii Excessive hypothermia

Combinations of these may be especially dangerous

- b SECONDARY CARDIAC FAILURE—Due to hypoxia of the myocardium or to inadequate coronary circulation consequent on coronary atheroma, haemorrhage, shock, hypotension.

SIGNS OF CIRCULATORY COLLAPSE—Pallor, weak or absent pulse. If carotid pulse cannot be felt the surgeon should be asked to palpate the aorta if the abdomen is opened or to apply his ear to the precordium if it is not. Respiratory arrest dilatation of the pupils.

TREATMENT OF CIRCULATORY COLLAPSE\*—This may be impossible in the presence of cardiac pathology, continuing hypoxia, low blood volume or brain trauma. Early diagnosis both of failure of the circulation and of its exact nature is imperative if the patient is to survive. Recovery is unlikely if artificial circulation is not re-established in five minutes. Owing to damage of cerebral cortex by the hypoxia, cases may recover but show signs of cerebral deterioration. Many such cases recover only to die within a few hours without regaining consciousness.

- 1 Lower head of table to increase cerebral circulation. Stop the operation.
- 2 See that adequate interchange of gases is instituted and maintained throughout subsequent proceedings. Do not waste time over: (a) Endotracheal intubation, this can be done later when the condition improves. (b) Injecting adrenaline into the heart. (c) Giving intra-arterial transfusions. (d) Aseptic technique.

If the circulation has ceased as suggested by: (a) Absence of pulsation of a visceral artery. (b) A silent precordium. (c) A dilated pupil. (d) Absence of bleeding from the incision, the surgeon should be asked to initiate artificial circulation by cardiac compression immediately.

- 3 CARDIAC PUMPING—This was first done in animals by Schiff of Geneva in 1874 and in man by Igelsrud in 1901. Electrical defibrillation came later, the first successful case in man being by Beck of Cleveland in 1947. The first successful cardiac massage in England was by Arbuthnot Lane of Guy's Hospital in 1902 at the suggestion of Ernest Starling.

a Transthoracic Route—This is the method of choice as it is more efficient than the abdominal approach and also allows

*Treatment of Circulatory Collapse continued*

electrical defibrillation to take place should it be necessary. An incision large enough to admit the hand is made in the fourth or fifth left intercostal space stopping 1 in from the sternum to avoid dividing the internal mammary artery and extending to the mid axillary line. The left arm of the patient should be abducted. To start with the heart and the pericardium should be lifted up against the sternum and compressed against it 50-60 times per minute. Later if necessary the pericardium should be incised. Care must be taken not to rupture the flabby myocardium by the finger tips. A rib retractor may be necessary after a minute or two to ease the pressure on the surgeon's wrists. Time must be allowed for the refilling of the heart after each compression. Elevation of the legs and intra-venous infusion will encourage filling. When oxygenated blood has been squeezed from the heart into the aorta for a minute or two the situation is reviewed and the diagnosis made of asystole or ventricular fibrillation the former being more common.

*Further Treatment of Asystole*—This is not standardized but the following measures have been suggested: (i) Continue cardiac compression until a normal beat returns. (ii) If the returning beat is weak stimulate the myocardium by injection into the left auricle—or the thoracic aorta proximal to a clamp applied to divert all the circulating blood towards the brain—of 3-5 ml of a mixture of noradrenaline in saline (1 ml of 1-1000 solution to 10 ml)\*. (iii) If the response is still unsatisfactory 3-4 ml of 10 per cent calcium chloride should be similarly injected to restore the cardiac tone. These injections may initiate ventricular fibrillation which must be appropriately treated. Pumping must be continued for a long period.

*Further Treatment of Fibrillation*—After the tone of the heart muscle has been improved by compression for several minutes electrical defibrillation must be attempted. The passage of an electrical current through the heart causes all its muscular fibres to have their refractory period simultaneously. A normal beat is then stimulated by manual compression. Two spoon shaped electrodes are applied to the heart. They should be covered by gauze soaked in saline to prevent burning. One shock may convert the fibrillation to asystole or a succession of shocks at intervals of 0.5 sec may be required at voltages increasing up to 250 v†. The operating team should wear rubber gloves to protect themselves from electric shock. If electrical methods are unavailable or unsuccessful 5 ml of 4 per cent potassium chloride or

McMillan, I. K. R. *Cockett, F. B. and Styles, P., Thorax* 1952 7 25

† McMillan, I. K. R. *Brit med Bull.* 1955 11 3 [229]



**Circulatory Collapse—After Treatment** *continued*

In the case of a death on the operating table legal responsibility will exist only if either the surgeon or the anæsthetist has failed in the proper execution of his functions as a person of professional skill. The anæsthetist no more than the surgeon is legally responsible if death has resulted from a genuine error of judgement but he is expected to exercise care skill and judgement in all that has to do with the anæsthetic procedure including the assessment of the patient's fitness to withstand the strain of anæsthesia successfully. The actual decision to operate together with the choice of operation is the responsibility of the surgeon.

- 12 Hypertension**—This may be due to mild respiratory obstruction in its early stages to hypercapnia to over vigorous transfusion to abuse of pressor drugs or to an unsuspected phæochromocytoma.

## CHAPTER X

GASES USED IN ASSOCIATION  
WITH ANÆSTHESIAOXYGEN [O<sub>2</sub>]

Discovered in 1771 by Priestley and by Scheele at approximately the same time. Modern use popularized by J. S. Haldane during the first world war. First prepared commercially by Linde in 1895 by the fractional distillation of air.

**Preparation**—

- 1 The fractional distillation of liquid air prepared by pressure and heat abstraction (Linde)
- 2 The electrolysis of water
- 3 The Le Brin process of heating barium oxide (BaO) to 500 C so forming barium peroxide BaO<sub>2</sub>, which is heated still more—to 800 C—when it parts with oxygen becoming barium oxide again.

Oxygen is supplied in cylinders at a pressure of 120 atmospheres (1800 lb per sq in). In the cylinder it is in the gaseous state.

**Properties**—Molecular weight 32. Solubility in water at 37 C 2.4 volumes per cent. in water at 0 C 4.9 volumes per cent. Specific gravity 1.105 (air is 1.000). Electric sparks convert it into ozone (O<sub>3</sub>).

With oil or grease oxygen under high pressure will cause an explosion. Oxygen (and nitrous oxide) cylinders should be turned on outside the operating theatre brought in faintly hissing so that when reducing valve is connected pressure is not built up in it suddenly but gradually. At other times when the cylinders are turned on the flow meters should themselves be on. It is rapidly absorbed from the alveoli this may be an argument for leaving

an inert gas (nitrogen or helium) in the lungs after operation in order to prevent atelectasis after complete absorption of oxygen. Oxygen diffuses from an alveolus whose bronchus is blocked in 15 minutes. Nitrogen diffuses in 16 hours.

**Types of Oxygen Lack.**—Oxygen lack not only stops the machine but wrecks the machinery (J S Haldane). Hypoxia or anoxia is oxygen lack in tissues. Anoxæmia is oxygen lack in the blood.

HYPOXIA	OXYGEN CAPACITY	ARTERIAL OXYGEN		VENOUS OXYGEN		ARTERIOVENOUS DIFFERENCE
		Content	Tension	Content	Tension	
Hypoxic	Normal	Decreased	Decreased	Decreased	Decreased	Decreased
Anæmic	Decreased	Decreased	Normal	Decreased	Decreased	Decreased
Stagnant	Normal	Normal	Normal	Decreased	Decreased	Increased
Histotoxic	Normal	Normal	Normal	Increased	Increased	Decreased

Modified from Myer Saklad.

The solubility of oxygen in arterial blood in a patient breathing air is 0.3 ml per 100 ml. One gramme of hæmoglobin can combine with 1.34 ml of oxygen. At a  $pO_2$  of 100 mm Hg hæmoglobin is 97.5 per cent saturated and a normal patient with 15.6 g of hæmoglobin per 100 ml of whole blood carries 20.4 ml of oxygen per 100 ml of whole blood in combination with hæmoglobin.

Reduced utilization of oxygen by tissues may be of the following types: (1) Hypoxic, (2) Anæmic, (3) Stagnant, (4) Histotoxic. The first three were described by Barcroft (1909), the fourth by Peters and Van Slyke (1931).

1. **HYPOXIC HYPOXIA**—Occurs when partial pressure of oxygen in blood is reduced. Saklad further subdivided this group into (a) atmospheric, (b) tidal, (c) alveolar. Seen in anaesthesia when oxygen tension of lung gases is deliberately cut down when airway is obstructed, respiratory muscles or respiratory centre are depressed, when there is defective pulmonary absorption, e.g. in emphysema, atelectasis, pulmonary oedema, presence of alveolar mucus, pneumothorax, hydrothorax, increased intra-abdominal pressure splinting the diaphragm, diffuse pulmonary hæmorrhages in blast injury. It is the type seen at high altitudes and in congenital heart disease with shunting of venous blood to left side of heart and conditions in which blood is circulating through unaerated alveoli. It is present during internal paradoxical respiration when there is an open pneumothorax.

The oxygen saturation of the arterial blood is reduced from its normal of 95 per cent while the oxygen content normally 19.5 vol per cent is also reduced.

Types of Oxygen Lack *continued*

- 2 **ANÆMIC HYPOXIA** (hæmoglobic hypoxia) —The oxygen content is reduced. Oxygen carrying capacity of blood is reduced in proportion to degree of anæmia although the blood-oxygen tension ( $pO_2$ ) is normal. Also in carbon monoxide poisoning.
- 3 **STAGNANT HYPOXIA** —Occurs when circulation is slowed so that in spite of adequate oxygen saturation and oxygen tension of arterial blood each red cell gives up a larger part of its oxygen owing to its longer stay in the capillaries. The oxygen content of venous blood is reduced. Typically seen in surgical shock and cardiac failure in peripheral vascular disease cerebral and coronary thrombosis etc. Can occur in local areas such as finger nails owing to cold seen in obstruction to venous return of skin of face and forehead produced by anæsthetic harness.
- 4 **HISTOTOXIC HYPOXIA** —Occurs when tissues are unable to utilize the normal supply of oxygen brought to them seen in cyanide poisoning and overdosage of narcotics and anæsthetics and when there is œdema of a tissue œdema interfering with diffusion of oxygen from capillaries to tissue cells. The arterial oxygen content and tension are normal and the venous oxygen saturation is higher than normal.

Demand hypoxia is seen in hyperpyrexia and hyperthyroidism.

Diffusion hypoxia\* may be seen during recovery from nitrous-oxide anæsthesia when the gas may diffuse into the alveoli in such volumes as to account for 12 per cent of the expired atmosphere this may so dilute the inspired air as to seriously reduce the oxygen saturation and so may prove dangerous to a handicapped patient e.g. after pneumonectomy in coronary disease where there is residual respiratory depression. The remedy is to give oxygen in high concentration towards the end of the anæsthesia and for some time after its termination.

**Diagnosis of Hypoxia** —The scientific way of doing this is by the use of an oximeter which assesses blood oxygen saturation. Such instruments as that of Milliken and its adaptations are attached to the lobe of the ear †

**THE EFFECTS OF OXYGEN WANT** —There is considerable individual variation in this.

- 1 **THE RESPIRATORY SYSTEM** —Hyperpnœa which only appears if the oxygen percentage is below 16 per cent it varies with individuals and is due to reflex stimulation of respiratory centre by chemoreceptors in aortic and carotid bodies which react to the lowered oxygen tension but not to a lowered oxygen content (as seen in anæmia and in carbon monoxide poisoning). The respiratory centre becomes less sensitive to carbon dioxide with increasing hypoxia. Dyspnœa and hyperpnœa are not necessarily indications for oxygen therapy as both may be seen without hypoxia just as hypoxia can occur without these symptoms.

Fink, B. R., *Anæsthesiology* 1955 16 511

† Stephen, C. R. Slater H. M. Johnson, A. L. and Sekelj, P. *Ibid* 195 12 541

- 2 **THE CARDIOVASCULAR SYSTEM**—The coronary and cerebral vessels dilate blood pressure and pulse rate are increased at first later they both decrease—the so-called hypoxic pressor and depressor phases. The E.C.G. T wave becomes inverted or decreased and there is slowing of conduction and a lengthening of the P-R interval. Capillaries lose their tone and their walls allow the leakage of fluid and cells into the tissues.
  - 3 **THE CENTRAL NERVOUS SYSTEM**—The nervous tissue is more susceptible to oxygen want than any tissue in the body. The blood flow to the brain is increased an effect also produced by the raised carbon-dioxide tension which is often concurrent. Later oedema of the brain results from capillary damage. The C.S.F. pressure is increased. Vomiting may be seen. Following acute or prolonged chronic hypoxia the following may be seen: (a) Acute psychoses (b) Increased psychomotor activity (c) Decerebrate states (d) Psychoneurotic states (e) Chronic psychotic states (f) Parkinsonism (g) Blindness or visual agnosia.
  - 4 **THE LIVER**—Inability to deaminate amino acids damage to cells. Vasodepressor material (V.D.M.) formed in liver and muscles which interferes with capillary tone is destroyed in the presence of oxygen by vasoexcitator material (V.E.M.) formed in kidneys. V.D.M. accumulates during periods of hypoxia. The blood-sugar is raised.
  - 5 **THE KIDNEYS**—Hypoxia tolerated fairly well.
- Hypoxia can be suspected if the pulse rate slows down ten or more beats within a few minutes of the administration of 100 per cent oxygen.

McDowell† has shown that the harmful effects of oxygen lack are due to the upset of electrolytic balance between the inside and outside of cells upon which their excitability apparently depends. The sodium chloride within the tissue cells is very much less than the sodium chloride in the intercellular fluid and plasma. The sodium pump is the name given to the mechanism which prevents the entrance of sodium chloride into the cells. Oxygen lack makes the cell membranes more permeable and so upsets the balance and allows sodium chloride to enter the cells.

#### **Causes of Hypoxia during Anaesthesia.—**

- 1 Low oxygen percentage in inspired atmosphere
- 2 Respiratory obstruction
- 3 Deficient tidal exchange which causes dead space gases to be moved to and fro along gas tubes
- 4 Delayed diffusion in lungs because of abnormality of pulmonary epithelium or to mucus overlying it
- 5 Shock and hæmorrhage

Hypoxia depresses respiratory centre and if not great stimulates the chemoreceptors of the carotid and aortic bodies with production of increase in rate and depth of breathing.



**Oxygen—Causes of Hypoxia during Anæsthesia** *continued*

This secondary stimulation disappears with the onset of gross hypoxia

**Post mortem Changes caused by Hypoxia**—Brain Punctate hæmorrhages in cortex and venous congestion in pia and basal ganglia Microscopic changes in cells of cortex and basal ganglia Necrosis of the third layer of the cerebral cortex Purkinje cells striate body and globus pallidus necrosis of the thalamus Hæmorrhages into epicardium and aorta systemic and pulmonary venous congestion congestion of submucosa of stomach and gut kidneys liver

**Oxygen Therapy**—Suggested by John Hunter in management of resuscitation in 1776 Given to asphyxiated neonates by Chaussier in 1780 Started by Beddoes at Bristol in 1794 when he was associated with Sir Humphrey Davy and James Watt Popularized by Haldane and Barcroft in 1917 By Yandell Henderson in the U.S.A.

Histotoxic hypoxia is probably not benefited by oxygen therapy

When oxygen percentage in inspired air is increased hæmoglobin becomes more saturated and the oxygen carried in solution in the plasma is increased Oxygen is carried in the blood (1) By physical solution (2) In combination with hæmoglobin At a  $pO_2$  of 100 mm Hg the normal tension 100 ml of blood carries 0.3 ml of oxygen in solution and 20.4 ml of oxygen combined with hæmoglobin At a  $pO_2$  of 160 mm Hg (complete saturation) or above the amount of oxygen carried by hæmoglobin only increases 0.5 ml per 100 ml of blood but the amount carried in simple solution increases in direct proportion to the  $pO_2$  so when pure oxygen is inhaled the total oxygen content of the blood is 2.2 ml per 100 ml an increase of over 10 per cent and the venous oxygen saturation rises from 75 per cent to 88 per cent Increasing the tension of oxygen in the alveolar air increases the coefficient of diffusion of oxygen so that more diffuses through abnormal pulmonary epithelium which may be present in bronchitis pneumonia pulmonary oedema etc

High oxygen tensions must not be given for more than a few hours continuously After several hours breathing 100 per cent oxygen there may be reduced vital capacity paræsthesiæ in limbs joint pains nausea and vomiting and retrosternal pain

In asthma or respiratory obstruction helium added to the inspired gases enables more oxygen to reach the alveoli and less effort to be expended on it It is evident from the shape of the oxygen dissociation curve that in many cases a large increase in the blood-oxygen saturation can be attained by relatively slight increase in the oxygen inspired especially if the patient is hypoxic before the administration is commenced

**THE EFFECTS OF INHALATION OF 100 PER CENT OXYGEN**

**Nitrogen** is eliminated from the lungs in two minutes and from the body in about two hours

**Carbon Dioxide**—As reduced hæmoglobin aids in the transport of carbon dioxide inhalation of 100 per cent

the amount of reduced haemoglobin interferes with the transport of carbon dioxide especially if the gas is given at a raised pressure. At 3 atmospheres enough oxygen is carried in solution (6 ml per cent—body requirements are 5 ml) to satisfy total body needs so no haemoglobin becomes reduced.

*Respiration*—This is often slightly depressed at first owing to the removal of the stimulating effect through chemoreceptors. Later there may be some stimulation because oxygen stimulates the mucosa of the lower respiratory tract and by dilating pulmonary capillaries causes slight pulmonary congestion and hence respiratory stimulation.

*Circulation*—There is decrease in the pulse rate from chemoreceptor effect. Cardiac output is lessened owing to effect both on rate and stroke volume. Slight increase in diastolic blood pressure. Blood vessels directly constricted reflexly via chemoreceptors dilated—former effect predominating. Cerebral vessels constrict coronary vessels also constrict but pulmonary artery dilates constricting in hypoxia. Very prolonged administration of oxygen may interfere with red-cell formation.

*Physical Effects*—There may be rapid absorption of gas from the alveoli leading to atelectasis from the middle ear leading to retraction of the drum if there is in addition obstruction of the Eustachian tube from the nasal sinuses causing headache.

#### ADVERSE EFFECTS OF HIGH OXYGEN ATMOSPHERES—

May occur in —

- 1 CHRONIC LARYNGEAL OBSTRUCTION—When symptoms of temporary cerebral depression may follow from sudden reoxygenation of arterial blood.
- 2 CHRONIC COR PULMONALE—E.g. emphysema. Reactions are usually confined to patients with a raised carbon dioxide tension in blood. Somnolence and coma may follow the reasons for the change being —
  - a The secondary result of chemoreceptor stimulation by hypoxia is stimulation of the cerebral cortex and removal of this by oxygen leads to cerebral depression.
  - b Removal of the hypoxic drive via the chemoreceptors may depress respiratory centre causing hypercarbia and depression.
  - c Neurological effects due to sudden increase in the pressure of the cerebrospinal fluid.
  - d Cerebral vasoconstriction due to oxygen.

To avoid carbon dioxide poisoning assisted breathing should accompany the early stages of oxygen therapy in chronic emphysema and in other conditions oxygen should be given intermittently and in gradually increasing tensions. When a patient with chronic emphysema or similar complaint is to be anaesthetized the immediate administration of high

**Oxygen—Causes of Hypoxia during Anæsthesia continued**

This secondary stimulation disappears with the onset of gross hypoxia

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**Carbon Dioxide**—As reduced hæmoglobin aids in the transport of carbon dioxide inhalation of 100 per cent oxygen by lessening

cent oxygen reduces the nitrogen tension in the blood so that the molecules of gas in the tissues diffuse into the blood and are carried away.

- 5 When metabolic rate is raised e.g. in post-operative thyrotoxicosis and hyperthermia because in these conditions the demand for oxygen is increased.
- 6 In all patients with liver damage in the immediate post-operative period.



F 34—Tudor Edward spectacle catheter carrier (British Oxygen Gases Ltd)

- 7 In carbon monoxide poisoning a high blood oxygen tension aids dissociation of carbon monoxide from haemoglobin. The only instance where oxygen therapy is effective in anaemic hypoxia.
- 8 In cases of severe headache due to retained intracranial air following encephalography. In migraine to produce vasoconstriction of cerebral vessels. Commercial oxygen is pure enough for inhalation and is much cheaper than medicinal oxygen. Oxygen therapy presents a definite fire hazard. In all cases it is important to see that a patent airway exists.
- 9 To assist denitrogenation before pure nitrous oxide-oxygen anaesthesia.

**MEASUREMENT**—This can be effected by—

- 1 A McKesson oxygen therapy apparatus from which Minnitt derived his original gas-air machine.
- 2 The Cowan and Mitchell Injector (1942). The air entrainment duct is covered by a rotating disk which has holes of varying size drilled in it the size of the hole governing the

**Oxygen Therapy continued**

oxygen atmospheres as with ether or cyclopropane is not always wise

- 3 **OXYGEN POISONING**—The prolonged inhalation of over 70 per cent oxygen may cause substernal distress reduction in vital capacity paræsthesias joint pains anorexia nausea and vomiting

The oxygen paradox was first described by Ruff and Strughold in 1939 and has recently been described by Latham\*. It is a temporary blackout due to the sudden administration of a high oxygen atmosphere seen in airmen. If the gas is first inhaled at low tensions and later gradually increased all effects are not seen

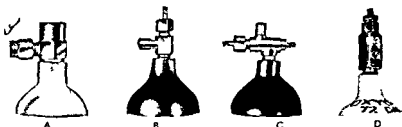


Fig 33—Type of cylinder outlet valves. A Bull nosed B Straight (Type 7) C Angled (Type 8) D American. (British Oxygen Gases Ltd)

- A high oxygen tension given to premature babies should be reduced gradually otherwise retrolental fibroplasia may occur. This condition can be treated by subjecting the infant to high oxygen tensions for a long time gradually reducing the tension to normal. Premature babies should only receive oxygen if they are cyanosed and then no more than 40 per cent concentration for as short a time as possible.

While in a healthy person inspiration of 100 per cent oxygen may be harmful if continued for more than a few hours 60 per cent can be inhaled indefinitely with impunity

**INDICATIONS**—The relief of all forms of hypoxia other than histotoxic

- 1 Cyanosis of recent origin. Acute heart failure acute pulmonary œdema (should be given under positive pressure of 4 cm of water) pneumonia atelectasis pulmonary embolism etc. Venous to arterial shunts intracardiac or intrapulmonary are the only types of hypoxæmia not completely corrected by the inhalation of 100 per cent oxygen
- 2 Following chest wounds or operations
- 3 In shock and severe hæmorrhage and coronary occlusion
- 4 To decompress distended bowels reduce surgical emphysema pneumothorax air embolism. The gas imprisoned in these cases is largely nitrogen. Prolonged inhalation of 100 per

With Model I the following data apply —

Oxygen Flow (litres Per Minute)	Number of Holes Open	Alveolar Air (Oxygen Percentage)	Carbon Dioxide in Bag (Percentage)
3	3	46	2.04
4	3	56	1.51
5	2	69	1.22
6	2	76	0.89
6	0	87	1.47
-	0	90	0.99
8	0	91	0.39

With Model II the percentage of oxygen in the alveolar air is 5% with an oxygen flow of 3 litres a minute. When 6 litres are flowing the percentage rises to 76. Thus with a B.L.B. mask alveolar oxygen percentage is raised six fold when 6 litres a minute of oxygen are flowing.

5 THE POLYTHENE DISPOSABLE MASK (Polymask Fig. 35)

6 AN OXYGEN CHAMBER OR TENT

Intravenous oxygen at the rate of 10 ml per minute is said to have antishock properties.

### CARBON DIOXIDE [CO<sub>2</sub>] *Carbon Dioxide*

Discovered by Von Helmont and isolated by Black in 1756. Became popular with anaesthetists soon after the work of Haggard and Vandell Henderson in the U.S. (1921) who recommended 5 per cent of the gas in oxygen and J.S. Haldane in Britain (1906). The gas should always be provided on anaesthetic machines.

**Properties** — Colourless odourless gas. Molecular weight 44. Specific gravity 1.500 (air is 1.000). Dissolves in water 100 volumes per cent at room temperature.

It is non flammable and is rapidly absorbed from the alveoli. It is stored in the liquid state in cylinders painted grey. The body forms about 200 c.c. per minute. Amount in atmosphere 0.04 per cent.

#### Preparation —

- 1 Action of heat on calcium carbonate
- 2 Action of acid on alkaline carbonate
- 3 During fermentation of grain in preparation of alcohol

#### Pharmacology —

**RESPIRATORY SYSTEM** — The respiratory centre is stimulated either specifically or owing to the acid properties of carbon dioxide in solution. Both rate and depth of breathing are increased. Respiration also reflexly stimulated by action of carbon dioxide on carotid and aortic bodies. If respiratory centre is depressed by morphine or anaesthetic agents its power of responding to carbon dioxide is lessened. An increase in the partial pressure in the blood of 1.5 mm. Hg (2 per cent) increases the tidal exchange 100 per cent. A decrease of 1.5 mm. Hg produces apnoea if stimuli from the periphery are damped down. Five per cent carbon dioxide is tolerated but higher percentages cause distress, dyspnoea, headaches etc. Above 10 per cent

**Oxygen Therapy—Measurement continued**

volume of air mixed with oxygen Between 40 per cent and 100 per cent of oxygen can be given and is led with air to a reservoir bag with non return valves No rebreathing takes place

- 3 Any simple water sight flow meter
- 4 An anæsthetic machine
- 5 Dial flow meters



Fig. 35.—The Plymouth (British Oxygen Co. Ltd.)

**TECHNIQUE**—Oxygen is led from a cylinder via a reducing valve and a flow meter and humidifier to —

- 1 **A NASAL CATHETER**—Size 9 is suitable and its terminal 3–4 in. should be smeared with analgesic cream. The distal end lies in the nasopharynx. With it an oxygen flow of 3 litres a minute raises the alveolar oxygen from 14 per cent to 27 per cent. A flow of 5 litres per minute, which is uncomfortable for the conscious patient, raises the alveolar oxygen to 38 per cent.
- 2 **A Y tube** made of glass with two bicycle valve tubes entering the nasopharynx. A flow of 6 litres per minute gives 42 per cent alveolar oxygen.
- 3 **TUDOR EDWARDS'S SPECTACLE FRAME CATHETER CARRIER** (Fig. 34). If air swallowing occurs the catheter is too far in.
- 4 **B.L.B. MASK**—The oronasal and the nasal types. Two sizes in each type. The oxygen flow must be sufficient to prevent collapse of the bag at the end of inspiration.

collapse of circulation) If pushed convulsions and twitchings occur

Look on hypercapnia during anaesthesia as a sin! Be against it! (Merriman) It may cause cardiac arrhythmia and by increasing the level of the blood potassium cardiac arrest hypertension vascular accidents including cyclopropane shock and self-perpetuating respiratory depression (Scurr) A tracheal tug may be a sign of hypercapnia Death in the period soon after operation may be due to hypercapnia there may be no post mortem findings to prove this

With the patient breathing adequate oxygen i.e. at least 5 per cent the fact that the colour is good does not prove that carbon dioxide is being properly eliminated A degree of hypoventilation sufficient to double the carbon-dioxide tension to 80 mm Hg reduces the alveolar oxygen tension to 54 mm Hg which will give about 80 per cent saturation of arterial blood or just about the cyanosis threshold in the average patient In other words the tension of carbon dioxide may be doubled in alveolar air before cyanosis gives a warning of hypoventilation \*

**CIRCULATORY SYSTEM**—Carbon dioxide produces local vasodilatation in peripheral capillaries It also releases noradrenaline Centrally it causes vasoconstriction later going on to vasodilatation Vagal action is potentiated by hypercapnia Cardiac rate and output increased Blood pressure raised—unless patient is in a state of sympathetic block as after high extradural analgesia The volume of blood flowing through the brain is increased up to 75 per cent if 5–7 per cent carbon dioxide is inhaled and this helps the stimulating effect of the gas on the respiratory centre Conversely a low blood-carbon-dioxide tension causes constriction of cerebral vessels lessens the cerebral circulation Moderate hyperventilation can reduce cerebral blood flow 35 per cent A rise in blood-carbon dioxide tension may cause arrhythmia The sudden change from hyper to hypocapnia may raise the plasma potassium level and so may cause arrhythmia or even ventricular fibrillation Anaesthesia does not protect against this

**CENTRAL NERVOUS SYSTEM**—Carbon dioxide excess causes depression headache nausea vertigo and later convulsions unconsciousness and anaesthesia It results in an increase in cerebral blood flow CSF pressure and brain volume

Muscular tone is increased and thus cardiac output is increased

Hypocapnia is a decreased tension of carbon dioxide in the blood It may produce apnoea and a decrease in capillary and venous tone cardiac output venous return and cerebral blood flow while the oxygen dissociation curve may be shifted to the left (i.e. oxygen saturation may be adequate but oxygen cannot be delivered to the tissues) but these changes tend to be theoretical Hyperventilation may cause a change in the pH of blood and a pale cold and perhaps lilac coloured skin of no clinical importance On the other hand it is



Carbon Dioxide—Pharmacology *continued*

the narcotic effect becomes more marked while at 30 per cent there is anæsthesia with no increased breathing rate or depth. At 40 per cent breathing is depressed apnoea results when the tension in alveolar air and blood is 110 mm Hg. This so-called carbon dioxide reversal may occur at concentrations of even 5 per cent if respiratory centre is deeply depressed by narcotics or hypoxia. Thus carbon dioxide should not be used for resuscitation in such cases.

## EFFECTS OF CARBON DIOXIDE INHALATION (HALDANE) —

Carbon Dioxide Percentage	Respiratory Volume	Percentage of Subject's Normal Respiratory Volume	Alveolar Carbon Dioxide Percentage
0.04 (air)	673	100	5.6
2.2	864	153	5.6
3.07	1216	226	5.5
5.14	1771	498	5.2
6.02	2104	857	6.6

**Hypercapnia** — There is always a tendency in anæsthesia for carbon dioxide accumulation to occur. An oxygen rich atmosphere by abolishing the respiratory drive and by effects on the carbon dioxide carriage by the blood will tend to cause hypercapnia. In patients suffering from chronic respiratory acidosis oxygen administration by removing the respiratory drive of hypoxia on the aortico carotid bodies can lead to carbon dioxide narcosis. Hypercapnia is one cause of respiratory depression occurring after anæsthesia. It should be treated by vigorous hyperventilation through a non rebreathing or an efficient closed-circuit with soda lime. In the conscious state the blood pH is controlled very carefully by the altering sensitivity of the respiratory centre but during anæsthesia with central and peripheral respiratory depression this adjustment is lost. By using intermittent positive pressure respiration during anæsthesia a normal acid base balance can be maintained but a minute volume twice that in the resting state is necessary perhaps because the hemodynamics of the pulmonary circulation are altered by the increased positive pressure used in ventilation. The prone lateral and head-down positions make the condition worse. Hiccups during anæsthesia is often associated with a respiratory acidosis while in this state more thiopentone is required to keep the patient quiet and he will have correspondingly increased post operative drowsiness. If necessary the operation should be periodically interrupted to allow for bouts of hyperventilation.

Hypercapnia during anæsthesia produces respiratory stimulation followed by depression and gasping breathing. Blood pressure and pulse rate increased the former due to carotid and aortic body reflexes not direct stimulant action on vasomotor centre. They are later depressed with circulatory collapse from myocardial failure. (Not vasomotor failure seen with hypoxic

a significant rise in its concentration in the body. In an anesthetized or narcotized patient with depression of the respiratory centre even 5 per cent carbon dioxide in oxygen will cause increased narcosis.

The gas is in fact, both a brain depressant and a respiratory stimulant in which the anæsthetic effect increases as the respiratory stimulating effect decreases. In the treatment of respiratory depression it is almost always contra-indicated.

**USE IN CHRONIC LARYNGEAL OBSTRUCTION**—In this condition the retention of carbon dioxide accustoms the respiratory centre to a high tension of the gas. When obstruction is relieved apnoea may follow unless carbon dioxide is added to the inspired gases to be gradually reduced. Another method of avoiding apnoea is to relieve the obstruction gradually as by partially occluding the tracheotomy tube temporarily.

**USE IN RESPIRATORY OBSTRUCTION**—May be dangerous as the combination of the resulting hyperpnoea with respiratory obstruction may produce an increased negative intrathoracic pressure and onset of pulmonary oedema.

The role of carbon dioxide in carbon monoxide poisoning is as yet unsettled (see Marriott H I *Brit med J* 1956 2 664—correspondence).

## NITROGEN [N<sub>2</sub>N]

First isolated by Rutherford in 1772

### Preparation —

- 1 Fractional distillation of liquid air
- 2 Heating ammonium nitrite

**Properties**—Molecular weight 28. Specific gravity 967 (air is 1000). Solubility in water and plasma at 37°C 1.28 volumes per cent.

It is chemically inert and will not support combustion or combine with water but is dissolved in body tissues from which it is displaced slowly. Solution in plasma is the sole method of transport of nitrogen in blood unlike oxygen which combines with hæmoglobin and carbon dioxide which forms carbamino compounds and combined with base is carried as bicarbonate. This accounts for the slow elimination of nitrogen. The elimination of nitrogen from the lungs breathing pure oxygen from a semiclosed inhaler at a flow rate equal to or greater than the minute volume and with no rebreathing takes about 2½ min. With a flow rate less than the minute volume the time for elimination is proportionately increased. With all nitrogen eliminated the refilling time equals the desaturation time if the patient breathes air. Once desaturation with nitrogen from the lungs has taken place a patient breathing from a closed system with oxygen supplied at basal rates requires more than 30 min to excrete nitrogen from the tissues\*. This is important in nitrous oxide-oxygen anaesthesia when the breathing bag should be periodically emptied to get rid of the nitrogen accumulating there from the tissues. 1.28 ml dissolves in 100 ml

**Carbon Dioxide—Hypercapnia—Central Nervous System continued**

possible that hyperventilation by causing cerebral vasoconstriction may result in harmful cerebral hypoxia\*. In practice the increased alkalinity resulting from hyperventilation probably does not interfere greatly with the dissociation of oxygen from oxyhaemoglobin i.e. the curve is not shifted greatly to the left

**Carbon Dioxide Estimation in Expired Air**—To collect a sample of end expiratory air a polythene catheter is inserted far down into the trachea connected to a syringe and a sample collected at the end of expiration. The average of several estimations is taken and fairly closely represents the alveolar air-carbon-dioxide tension. In health this closely corresponds to the carbon-dioxide tension in arterial blood.

The gas mixture to be examined is brought into equilibrium with a lightly buffered solution of sodium bicarbonate. The tension of carbon dioxide in the gas mixture determines the pH in the buffered solution and the pH in turn determines the colour of a suitable indicator †

**Carbon Dioxide in Anæsthesia**—Inspired air contains 0.04 per cent. Expired air contains 4 per cent. Alveolar air contains 5-6 per cent.

**USE DURING INDUCTION—**

- 1 To stimulate breathing after heavy premedication for a short time only i.e. to increase the tidal volume in the presence of a normally active respiratory centre
- 2 To expedite induction when volatile agents are used

**USE DURING MAINTENANCE—**

- 1 To increase depth of anæsthesia rapidly when volatile agents are in use. The addition of a little carbon dioxide at the first sign of returning reflex irritability—anæsthesia—may enable control to be re-established by increasing the patient's urge to breathe and so to inspire more of the anæsthetic agent. Its power to render the mucosa of the upper respiratory tract less sensitive to irritant vapours is here made use of.
- 2 To widen glottis either to overcome spasm or before blind intubation.

**USE DURING RECOVERY—**

- 1 To promote pulmonary ventilation and avoid collapse
- 2 To get rid of volatile anæsthetic agents from tissues via lungs. Beware of apnoea following deprivation of a high tension of carbon dioxide especially if respiratory centre is depressed by morphine cyclopropane pentothal etc.

**USE FOR RESUSCITATION**—If normal people inhale 12½ per cent carbon dioxide in oxygen unconsciousness will result in a short time. This is because a narcotic and not a stimulant effect is produced when the concentration in the inhaled atmosphere is such (7 per cent) that increased ventilation can no longer prevent

a significant rise in its concentration in the body. In an anaesthetized or narcotized patient with depression of the respiratory centre even 5 per cent carbon dioxide in oxygen will cause increased narcosis.

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- 2 Heating ammonium nitrite

**Properties**—Molecular weight 28. Specific gravity 967 (air is 1000). Solubility in water and plasma at 37° C. 1.28 volumes per cent.

It is chemically inert and will not support combustion or combine with water but is dissolved in body tissues from which it is displaced slowly. Solution in plasma is the sole method of transport of nitrogen in blood unlike oxygen which combines with haemoglobin and carbon dioxide which forms carbamino compounds and combined with base is carried as bicarbonate. This accounts for the slow elimination of nitrogen. The elimination of nitrogen from the lungs breathing pure oxygen from a semiclosed inhaler at a flow rate equal to or greater than the minute volume and with no rebreathing takes about 2½ min. With a flow rate less than the minute volume the time for elimination is proportionately increased. With all nitrogen eliminated the refilling time equals the desaturation time if the patient breathes air. Once desaturation with nitrogen from the lungs has taken place a patient breathing from a closed system with oxygen supplied at basal rates requires more than 30 min. to excrete nitrogen from the tissues\*. This is important in nitrous-oxide-oxygen anaesthesia when the breathing bag should be periodically emptied to get rid of the nitrogen accumulating there from the tissues. 1.28 ml dissolves in 100 ml

**Carbon Dioxide—Hypercapnia—Central Nervous System continued**

possible that hyperventilation by causing cerebral vasoconstriction may result in harmful cerebral hypoxia\*. In practice the increased alkalininity resulting from hyperventilation probably does not interfere greatly with the dissociation of oxygen from oxyhamoglobin i.e. the curve is not shifted greatly to the left

**Carbon Dioxide Estimation in Expired Air**—To collect a sample of end expiratory air a polythene catheter is inserted far down into the trachea connected to a syringe and a sample collected at the end of expiration. The average of several estimations is taken and fairly closely represents the alveolar air-carbon dioxide tension. In health this closely corresponds to the carbon dioxide tension in arterial blood.

The gas mixture to be examined is brought into equilibrium with a lightly buffered solution of sodium bicarbonate. The tension of carbon dioxide in the gas mixture determines the pH in the buffered solution and the pH in turn determines the colour of a suitable indicator†

**Carbon Dioxide in Anæsthesia**—Inspired air contains 0.04 per cent. Expired air contains 4 per cent. Alveolar air contains 5–6 per cent.

**USE DURING INDUCTION—**

- 1 To stimulate breathing after heavy premedication for a short time only i.e. to increase the tidal volume in the presence of a normally active respiratory centre
- 2 To expedite induction when volatile agents are used

**USE DURING MAINTENANCE—**

- 1 To increase depth of anæsthesia rapidly when volatile agents are in use. The addition of a little carbon dioxide at the first sign of returning reflex irritability—a sign of incomplete anæsthesia—may enable control to be re-established by increasing the patient's urge to breathe and so to inspire more of the anæsthetic agent. Its power to render the mucosa of the upper respiratory tract less sensitive to irritant vapours is here made use of
- 2 To widen glottis either to overcome spasm or before blind intubation

**USE DURING RECOVERY—**

- 1 To promote pulmonary ventilation and avoid collapse
- 2 To get rid of volatile anæsthetic agents from tissues via lungs. Beware of apnoea following deprivation of a high tension of carbon dioxide especially if respiratory centre is depressed by morphine cyclopropane pentothal etc.

**USE FOR RESUSCITATION**—If normal people inhale 12½ per cent carbon dioxide in oxygen unconsciousness will result in a short time. This is because a narcotic and not a stimulant effect is produced when the concentration in the inhaled atmosphere is such (7 per cent) that increased ventilation can no longer prevent

## CHAPTER VI

## ENDOTRACHEAL ANÆSTHESIA

- History.**—C.kite of Gravesend described oral and nasal intubation for resuscitation of the apparently drowned in 1788\*. Intubation from the neck, through a tracheotomy wound was performed in 1858 by John Snow in anæsthetizing animals. Trendelenburg used the method in man in 1871 occluding the trachea by an inflatable cuff.
- Macewen of Glasgow in 1878 passed a tube from the mouth into the trachea using his fingers as a guide. These early attempts were all made to prevent aspiration pneumonia in surgery of the upper air passages.
- Fritz Kuhn in 1901 extended the technique by using a flexible metal tube introduced on a curved guide through the mouth: his preference was for inhalation anæsthesia: the patient breathing to and fro along the tube.
- In 1907 Barthélemy and Dufour of Nancy, France, blew chloroform vapour and air from a Vernon Harcourt inhaler and a rubber catheter guided into the trachea by touch.
- Meltzer and Auer, physiologists working in the United States, pioneered insufflation endotracheal anæsthesia in animals in 1909. This entailed blowing an anæsthetic vapour at a positive pressure through a narrow tube into the trachea near the carina, the gases returning either through a second tube or alongside the insufflation tube. Elsberg and others in the same year applied the technique to man, while in 1912 Kelly of Liverpool brought the method to Britain.
- Hirstein of Berlin and Killian of Freiberg—the original bronchoscopist—pioneered direct laryngoscopy in 1896 and Chevalier Jackson published a book on the subject in 1907 and in the years following this popularized intubation. Jackson did his first bronchoscopy in 1899.
- As a result of their experiences during World War I, especially in plastic surgery, as anæsthetists to Sir Harold Gillies at the British Army Plastic Unit at Sidcup (1920) Magill and Rowbotham used first insufflation through two narrow tubes—one for leading gases in, the other for carrying them away—and later inhalation endotracheal methods. Magill published his results of blind nasal intubation with a single wide bore rubber tube during the years following 1928.
- Inflatable cuffs have been used for many years but were re-introduced by Waters and Guedel in 1928.
- In recent years the attitude to intubation has altered radically because

**Nitrogen—Properties continued**

of blood plasma The gas is five times as soluble in fat as in water

Nitrogen diffuses from an isolated lung lobule whose bronchus is tied in 16 hours Oxygen under similar conditions diffuses in 15 minutes

**HELIUM [He]**

Isolated by Ramsey in 1895

**Preparation** —From natural gas in the U S Air contains 1 part in 85 000

**Properties** —Inert colourless odourless gas Molecular and atomic weights 4 Specific gravity 178 (air is 1000)

When helium replaces the nitrogen of air the resulting mixture of helium and oxygen has a specific gravity of 341 (air is 1000) Because of its low density the gas will flow through an orifice three times as fast as air so in patients with partial respiratory obstruction 20 per cent of oxygen with 80 per cent of helium will enable more oxygen to get to the alveoli with the same effort or the same ventilation will take place with less effort than when air is inhaled Absorption rate from alveoli very slow a property made use of at the end of an anæsthetic when its introduction into the alveoli helps to prevent atelectasis Helium has a low coefficient of solubility and high rate of diffusion compared with nitrogen It decreases the resistance to breathing and so is used with oxygen in treatment of asthma and respiratory obstruction

If fed through a nitrous oxide flow meter the reading must be multiplied by 3.3 to get the approximate rate of flow in litres a minute

**XENON [Xe]**

First used in anæsthesia by Cullen and Gross in 1951 \*† Has a potency roughly equivalent to that of ethylene Is non flammable Causes no respiratory depression and no cardiac arrhythmia Biochemical change is not marked Different electroencephalographic changes are seen with xenon than with other inhalation agents ‡ Deep anæsthesia results from the inhalation of xenon at elevated pressures and hypoxia is not seen if the partial pressure of oxygen in the respired atmosphere is maintained at 200 mm Hg The amount of xenon in the arterial blood is directly proportional to the partial pressure of the gas in the inspired atmosphere

**OTHER GASES**

Argon is reported to have mild anæsthetic properties as also is sulphur hexafluoride §

Cullen, S. C. and Gross, E. G. *Science* 1951 **113** 58

† Morris, L. E. and others *Ibid* 1955 **16** 3

‡ Pittinger, C. B. Movers, J. Cullen, S. C. Fe therstone, R. and Gross, E. G.

*Anesthesiology* 1953 **14** 20.

§ Virtu, R. W., and Waver, R. H. *Ibid* 1952 **13** 605

with it the attached epiglottis so that the cords can be visualized. The superior aspect of the epiglottis is supplied by the 9th nerve the inferior (posterior) aspect by the internal laryngeal and hence as the inferior aspect of the epiglottis is not touched and thereby stimulated the Macintosh instrument can be used at a lighter plane of anesthesia than the orthodox pattern without producing laryngeal spasm. An additional advantage of the Macintosh instrument is that during intubation the tube does not hide the cords from view.

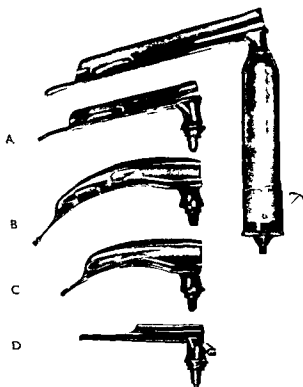


Fig. 36-7b. Reilly's laryngoscope showing types of detachable blade. A Magill B Macintosh C Macintosh (Small) D Shadwell (British Oxygen Gases Ltd.)

The following are some of the many modifications which are available.

The Kirk Rae laryngoscope\* consists of a single handle with several interchangeable blades requiring only quick rotation through an angle of 90° to secure or remove a blade (Fig. 36).



**History continued**

- 1 The use of muscle relaxants and especially the use of succinylmethonium has made intubation relatively easy quick and atraumatic
- 2 The use of muscle relaxants has greatly increased the need for intermittent positive pressure respiration and this is usually more satisfactorily carried out if the patient is first intubated

**Apparatus —**

**TUBES**—Those most commonly used are the wide bore Magill tubes of mineralized rubber. They can be used for either nasal or oral intubation the latter having thicker walls. The following are their sizes and their correspondence with the French catheter gauge (external diameter)

OO Magill	≈ 13 F C G	5 Magill	≈ 27 F C G
OA	≈ 16	6	≈ 29
O	≈ 17	7	≈ 30
1	≈ 18	8	≈ 32
2	≈ 20	9	≈ 34
3	≈ 23	10	≈ 37
4	≈ 25		

Other tubes are made of semi rigid material the Portex plastic tubes (polyvinyl chloride) being useful. Portex tubes if boiled and left to cool on a curved wire stylet will permanently take the curve of the wire. Another type of tube is made of a spiral coil of wire embedded in thin rubber to prevent kinking. These hexometallic tubes have been known to cause obstruction because of bubble formation in the rubber wall of the tube. Inflatable cuffs can be incorporated with the rubber tubes or loose ones can be slipped on using French chalk as a lubricant.

Tubes should be cleaned outside and inside with a test tube brush. Repeated boiling softens the rubber and soon ruins the tubes so sterilization should be by 1-1000 biniodide solution or by a solution containing 3 per cent of hexachlorophene which should remain in contact with the tube for at least 1 min. Magill tubes should be stored in a circular box 7 in in diameter so that their curve is maintained. Rubber tubes can be softened if they are soaked for 1 to 1½ hours in paraffin oil this will also make the tube larger a No 3 equalling a No 5 after soaking. A pen nib dipped in 50 per cent solution of silver nitrate can be used to write on tubes.

**LARYNGOSCOPES**—The prototype is that of Chevalier Jackson later modified by Magill and Palmer J. Flagg. The blade and handle may be either parallel or set at an angle one to the other the handle containing the 3 volt battery. Various types and sizes of blade are in use the Magill instrument being very popular. While the usual laryngoscope blade is designed to lift the epiglottis forward the Macintosh blade is shorter and its tip enters the vallecula lifts the base of the tongue and

**INFLATABLE CUFFS** (Figs 39-40) — Used to ensure airtight endotracheal anaesthesia instead of pharyngeal gauze packing. The Hewer pilot balloon shows the state of the cuff when it is inflated in the trachea. There is the danger when using a cuff of causing sloughing of the tracheal mucosa. The pressure in a cuff when comfortably inflated may be 120-180 mm Hg but this does not correspond to the pressure applied to the tracheal mucosa. Should sloughing happen bronchoscopic removal of sloughs may be required. Cuffs should not be inflated to a pressure greater than that needed to prevent audible leakage of gas when the reservoir bag is compressed. The integrity of the inflatable cuff must always be tested before use. Should gauze packing be used it must be well lubricated to prevent pharyngeal trauma. Failure to remove a gauze pack has resulted in many deaths.

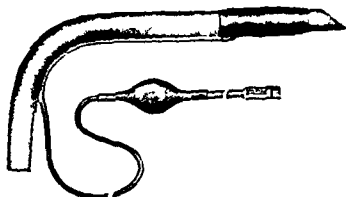


Fig. 4. The Orlonon k.k.g.e. lotr. h. l. tube (Medical and Industrial Equipment Ltd.)

Pinson points out that an airtight circuit results from firmly bandaging the lower jaw backwards so that the mouth is tied open. This places the base of the tongue in contact with the posterior pharyngeal wall. In edentulous patients Thornton's rubber mouth prop helps to get an airtight joint between mask and face. A moistened chin bandage inserted inside each cheek secures the same result.

As cuffs prevent leakage between the wall of the trachea and the outer wall of the tube they are useful in closed circuit anaesthesia. They also prevent blood, mucus and vomitus from entering the lungs and so are useful in intestinal obstruction with regurgitant vomiting and in operations on the upper air passages.

**LUBRICANTS** — To intubate atraumatically the tube and laryngoscope should be smeared with either a greasy or a water soluble lubricant. Paraffin deteriorates rubber and castor oil is preferable (Lunn R.W.). A local analgesic can be incorporated

Apparatus—Laryngoscopes *continued*

The Bowen Jackson laryngoscope\* is a modification of Macintosh's design. It has a rather long blade and the smallest possible step consistent with adequate tongue deflection. It occupies less room in the mouth than other instruments and is useful in difficult cases.

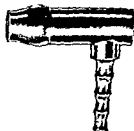


Fig. 37—Rowbotham's c-n-ecto  
(British Oxygen Gas Co. Ltd.)

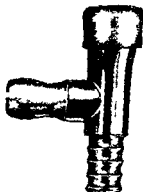


Fig. 38—McGill suction union  
(British Oxygen Gas Co. Ltd.)

**INTUBATING FORCEPS**—Magill's instrument is commonly used and is made in two sizes. Lundy's modification is a small bulb attached to the tip of one of the blades so that its illumination facilitates intubation.

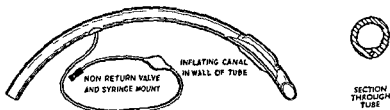


Fig. 39—Self-inflating endotracheal tube with inflatable cuff  
(British Oxygen Gas Co. Ltd.)

**ANGLE PIECES**—These connect the endotracheal tube to the breathing tube of the gas machine. Several designs are in use and they are made in various sizes. The Rowbotham type makes a tight and secure joint (Fig. 37). In other types there is an adjustable cap for insertion of a sucker tube for use in wet lung anaesthetics and other cases where suction is required (Fig. 38).

which contains 40 mg. cocaine. Apply it to the nasal mucosa and their patency by shrinking the mucosa. A mixture of 1 per cent. of Arthrocaïne 1 per cent. of tetracaine 5 per cent. of oil and 2 per cent. of nupercaine 2 per cent. of oil in air also used for the purpose of depressing the laryngeal reflexes. It is important not to poison the patient with these drugs.

An efficient atomizer such as that of Macintosh (Fig. 41) Magill Rowletham Multi-jet or the Wall is essential for the patient inhaling the vapour into his larynx during intubation. Spray is done either before or after induction of anaesthesia. More efficient analgesia is secured if in addition the parietal pleurae are treated with analgesic solution (p. 2 Chapter XVIII).

The Macintosh laryngeal spray (Fig. 41) carries the local anæsthetic solution right down on to the cord via a rubber tube inserted through the nares. It is also convenient for applying an analgesic spray to the inside of the larynx via a laryngoscope.

**Technique of Blind Nasal Intubation.** It is essential that extreme gentleness should characterize the whole proceeding.

1. Examine nares for patency by listening to the patient's breathing with each naris alternately occluded.
2. Spray nares and upper respiratory tract with 4 per cent. cocaine solution realizing that it is toxic.
3. Select the largest size of tube that experience suggests will pass atraumatically through the larger naris. For a big man size 10 for a small woman size 5-7. See that the lumen of the tube is clean and patent. On the curve of the tube depends the position of the patient's head. The greater the curve the more flexed should be the head during intubation. An average position of the head advised by Magill is that adopted when sniffing the morning air.
4. Induce anaesthesia and produce hyperpnea by allowing carbon dioxide to be inhaled for a few breaths. A flow of 1 litre a minute is usually sufficient. Insert a drop of oil into the selected naris and remove mask. The Macintosh laryngeal spray can be used at this point.
5. Insert the tube into the naris so that its convexity is directed to the patient's feet. Thrust the tube directly backwards not upwards. Movement of the bevel by rotation of the tube may be necessary to overcome resistance either at this stage or when the tube enters the nasopharynx from the nose.
6. Slightly elevate the lower jaw to lift the epiglottis away from the posterior pharyngeal wall. Occlude the opposite nostril so that all breathing is taking place through the tube. If the right naris is used the head should be inclined slightly to the right and vice versa.
7. As the tube is inserted the patient must be breathing actively. The ear is placed near the proximal end of the tube which is so advanced that the audible tubular breathing is maximal. In blind intubation the anaesthetist should be led by the ear. The anaesthetist's left hand may be able to move the larynx to meet the tube by manoeuvring the thyroid cartilage. The fingers often feel a slight snap as bevel passes between the cords.

Apparatus—Lubricants *continued*

such as amethocaine 1 per cent metycaine 2-5 per cent nupercaine 1-10 per cent xylocaine 2-4 per cent. Xylodase is useful for this purpose it contains 5 per cent lignocaine and 0.015 per cent hyaluronidase in a water miscible base. While petrol eum jelly is the better lubricant for blind nasal intubation its water-soluble substitutes will not cause pneumonitis if they reach the lungs. An analgesic containing lubricant will reduce the incidence of extubation spasm. A lubricant of tragacanth paste causes less deterioration of inflatable rubber cuffs than does a greasy lubricant which contains paraffin. To shrink the mucosa before nasal intubation eudrine is useful and contains 0.75 per cent ephedrine in an oily base with aromatics.

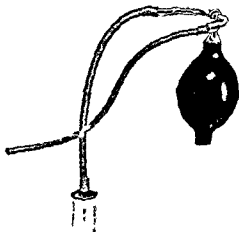


Fig. 41.—The McIntosh laryngoscope (B. H. O. Y. & Co. Ltd.)

The formula for Brennan's paste is —

Paraffin Dur	gr 15	Paraffin Moll. Alb	oz 1
Cer Alb	gr 30	Nupercaine Base	10 per cent

Soaking for five minutes in 1-1000 roccal solution kills most organisms which are likely to contaminate anaesthetic apparatus.

**Topical Analgesia** —The laryngeal reflex can be subdued if a suitable analgesic solution is sprayed on to the superior laryngeal aperture, the cords and the mucosa of the larynx and trachea. A German, Rosenberg, was the first to use a local analgesic (cocaine) to quieten the laryngeal reflexes (1895). Cocaine in 4-20 per cent solution is the most popular agent. It is said that the stronger solution is less rapidly absorbed because of the increased vasoconstriction that it causes, but the writer prefers the 4 per cent strength each ml of

which contains 40 mg. of cocaine. Applied to the nares it increases their patency by shrinking the mucosa covering the turbinates. Amethocaine 1 per cent, metycaine 5 per cent, xylcaine 2-4 per cent and nupercaine 2 per cent solutions are also useful for the purpose of depressing the laryngeal reflexes. It is important not to poison the patient with these drugs.

An efficient atomizer such as that of Macintosh (Fig. 41) Magill Rowbotham Multicaine or de Villiers is essential the patient inhaling the vapour into his larynx during inspiration. Spraying is done either before or after induction of anæsthesia. More efficient analgesia is secured if in addition the pyriform fossæ are treated with analgesic solution (see Chapter VIII).

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Technique of Blind Nasal Intubation *continued*

while there is usually some breath holding or cough except in deep anaesthesia. If the patient is at a light level of anaesthesia laryngeal spasm may be produced. It is often worth waiting for the expulsive cough that may follow spasm of the cords as the tube may slip between the cords which are widely abducted just before and after the cough.

8. Verify that the tube is in the trachea by the character of the breath sounds issuing from the proximal end of the tube. With a tube of reasonable length if breath sounds are free the distal end of the tube must be in the trachea. If the tube can be inserted to its full extent and no breath sounds can be heard it is probably in the oesophagus.

An experienced worker can usually intubate blindly following the intravenous injection of thiopentone (0.2-0.3 g) with suxamethonium (12-16 mg of active cation).

**DIFFICULTIES**—If the tube does not enter the trachea it should be pulled out far enough for its tip to be in the oropharynx before being thrust down again. A tube passed through the right naris comes to lie towards the left side of the glottis and vice versa. Should the tube not be in the trachea it may be situated—

- a. In the oesophagus i.e. posterior to its correct position either because the tube is not sufficiently curved or because the head is too flexed.
- b. On the anterior commissure of the larynx i.e. anterior to its correct position for reasons the opposite to those above.
- c. In the vallecula between the base of the tongue and the epiglottis. Rotation of the tube so that it slips down the lateral wall of the pharynx should overcome this obstruction.
- d. In one or other pyriform fossa lateral to its correct position. This is likely where the nose is asymmetric and is overcome by rotating the tube by moving the larynx laterally to meet the tube or by rotating the patient's head.
- e. Curled up in the pharynx. This will only occur with soft worn out tubes.

If unsuccessful blind intubation should not be persisted in or post operative local trauma may result. Tubes should always be handled daintily and not forcibly rammed down the patient's throat. If intubation through one naris is difficult it may be found to be quite simple through the other.

**THE CHOICE OF LIGHT OR DEEP ANAESTHESIA —****LIGHT ANAESTHESIA —***Advantages —*

- a. Less time required.
- b. For operation performed at a light plane of anaesthesia the patient gets less anaesthetic.
- c. Hyperpnoea easily produced and the explosive cough with wide cord abduction following breath holding may easily enable tube to enter trachea.

*Disadvantages —*

- a As available time is short the method calls for more experience and skill on the part of the anaesthetist
- b Spasm of the glottis may not be followed by a cough and so may hinder intubation

**DEEP ANÆSTHESIA —***Advantages —*

- a Absence of reflexes so that cough and spasm are absent
- b Plenty of time available for intubation
- c Direct intubation via the laryngoscope can be immediately proceeded with if necessary

*Disadvantages —*

- a Takes longer time for induction
- b May mean that depth of anaesthesia is greater for intubation than for operation

**Technique of Direct vision Nasal Intubation** — This is necessary if blind intubation fails and if a nasotracheal tube is desirable. It is the preferred method of nasotracheal intubation with intra-venous barbiturate and relaxant.

The laryngoscope is inserted after anaesthesia is induced as for direct orotracheal intubation. The tube tip having been inserted as far as the hypopharynx is guided between the cords either by slight movement of the tube or with the aid of intubation forceps. If the tube is too curved it may impinge against the anterior commissure of the larynx and this can often be remedied by keeping the tube pressed against the anterior commissure with drawing the laryngoscope flexing the head and pushing the tube home blind. Occasionally a tube cannot be guided successfully into the trachea from the nose it must then be withdrawn and inserted via the mouth.

**Technique of Direct vision Orotacheal Intubation** — Anaesthesia must be well established in the second plane or below and relaxation must be profound. Less harm is done by getting the patient a little too deep than by using force in a patient with active reflexes and rigid neck muscles. Absence of reflexes and adequate muscular relaxation are necessary for laryngoscopy in all but expert hands. Analgesia of the pharynx and larynx facilitates intubation by this route. The angle formed by the mouth with the pharynx must be converted into a straight line by lifting up the base of the tongue and the tip of the epiglottis.

Before induction the shape of the jaws and the condition of the teeth are carefully assessed. Loose or filled teeth especially upper incisors may be damaged by the blade of the laryngoscope.

*Difficulties may be expected in patients with —*

- 1 Short muscular necks and a full set of teeth
- 2 Receding lower jaws. Increased distance between the mental symphysis and the lower alveolar margin which requires wider depression of the lower jaw during intubation
- 3 Long high arched palates and a narrow deep mouth
- 4 Protruding upper incisors— rabbit teeth



Technique of Blind Nasal Intubation *continued*

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- b May mean that depth of anaesthesia is greater for intubation than for operation

**Technique of Direct vision Nasal Intubation** — This is necessary if blind intubation fails and if a nasotracheal tube is desirable. It is the preferred method of nasotracheal intubation with intravenous barbiturate and relaxant.

The laryngoscope is inserted after anaesthesia is induced as for direct orotracheal intubation. The tube tip having been inserted as far as the hypopharynx is guided between the cords either by slight movement of the tube or with the aid of intubation forceps. If the tube is too curved it may impinge against the anterior commissure of the larynx and this can often be remedied by keeping the tube pressed against the anterior commissure with drawing the laryngoscope flexing the head and pushing the tube home blind. Occasionally a tube cannot be guided successfully into the trachea from the nose it must then be withdrawn and inserted via the mouth.

**Technique of Direct vision Orotacheal Intubation** — Anaesthesia must be well established in the second plane or below and relaxation must be profound. Less harm is done by getting the patient a little too deep than by using force in a patient with active reflexes and rigid neck muscles. Absence of reflexes and adequate muscular relaxation are necessary for laryngoscopy in all but expert hands. Analgesia of the pharynx and larynx facilitates intubation by this route. The angle formed by the mouth with the pharynx must be converted into a straight line by lifting up the base of the tongue and the tip of the epiglottis.

Before induction the shape of the jaws and the condition of the teeth are carefully assessed. Loose or filled teeth especially upper incisors may be damaged by the blade of the laryngoscope.

Difficulty may be expected in patients with —

- 1 Short muscular necks and a full set of teeth
- 2 Receding lower jaws. Increased distance between the mental symphysis and the lower alveolar margin which requires wider depression of the lower jaw during intubation
- 3 Long high arched palates and a narrow deep mouth
- 4 Protruding upper incisors— rabbit teeth

Technique of Direct vision Orotracheal Intubation *continued*

- 5 Difficulty in opening the jaw as in multiple arthritis involving the temporomandibular joints and spondylitis of the cervical spine causing rigidity of the neck so that the head cannot be satisfactorily positioned trismus etc. Absence of upper incisors usually makes laryngoscopy relatively easy
- 6 Contractures of the tissues in the front of the neck from burns resulting in flexion of the neck

The mouth and pharynx usually at an angle are brought nearest to a straight line if the head is elevated 5-6 in. on a pillow and extended at the atlanto-occipital joint. This is the best position for laryngoscopy although Chevalier Jackson originally recommended that the head should be in full extension without a pillow. When the Macintosh laryngoscope is used the head should be flexed moderately on the neck and the blade of the instrument should be inserted towards the right side of the patient's mouth to prevent his tongue from blocking the view of the larynx.

With the patient properly anesthetized and in the proper position with a strip of adhesive protecting the upper incisor teeth the lubricated laryngoscope blade is gently inserted into the mouth and progressively advanced.

Landmarks are (1) The base of the tongue (2) The uvula (3) The epiglottis

When the epiglottis is seen it is elevated by the tip of the blade care being taken not to scratch the posterior pharyngeal wall. The tongue and epiglottis are now lifted forwards i.e. in the direction of the ceiling. The upper teeth must not be used as a fulcrum. As the curtain of the epiglottis is lifted forward the cords are exposed to view and are identified by their pallor. If the cords are obscured by spasm of the superior laryngeal aperture the aryepiglottic and ventricular folds the knobby appearance of the arytenoids gives a clue to their position these projections being behind the cords and nearer the anesthetist. Occasionally only the posterior part of the glottis can be visualized and then the tube must be inserted anterior to the arytenoids using a fully curved tube encasing a fully curved wire introducer. Even the expert sometimes fails to get a good view of the cords. Should the reflexes or muscular tone return during laryngoscopy the anesthesia must be deepened.

The blade can be either inserted in the middle line of the mouth or along its right side pushing the tongue to the left. With prominent teeth the latter route may be preferable.

When using the Macintosh instrument the blade is passed along the tongue to the vallecula and is tilted forwards so that the epiglottis is drawn away to reveal the cords. A tube with a curve is important a straight one being difficult to insert.

When the cords are visualized and sprayed with cocaine solution the lubricated tube is passed either down the laryngoscope or at the side of it into the larynx and trachea. With the Magill laryngoscope a large tube obscures the view of the cords. With the

tube may enter the œsophagus. As there may be apnoea the anaesthetist may not be sure as to the lie of the tube. This can be determined by lightly blowing down the tube. If it is in the trachea the chest will expand and the air will be heard issuing from the tube during the succeeding passive expiration. With the tube in the œsophagus chest expansion is not seen and there is evidence of the insufflated air bubbling down the œsophagus. Another method of differentiation is to push sharply on the chest when a short blast of air will be heard if the tube is in the trachea. Macintosh advises that if the mass of the tube hides the cords from view the tube should be threaded over a long gum-elastic catheter or curved bulbous ended wire which has previously been passed under direct vision and after intubation can be withdrawn. This method is also useful if the patient's anatomy makes a good view of the cords difficult. The reflexes should of course be inactivated by topical analgesia and/or by muscle relaxants.

If when visualized the cords are in spasm this must be allowed to relax before the tube is inserted. Forcing the cords is only justifiable in the presence of alarming hypoxia in patients whose condition under oxygen lack is particularly dangerous. If the cords are forced apart a round ended semi rigid tube well lubricated should be used. Once between the cords it can be instantly blown down with the rapid relief of the hypoxia.

With the tube in position the laryngoscope is withdrawn and a prop or gag is placed between the teeth to prevent the tube from being bitten.

If a cuff is to be used its inflation should be delayed until the patient has settled down again and has become deeper otherwise coughing and breath holding may be produced and delay the onset of adequate anaesthesia.

#### **Technique of Orotracheal Intubation without Laryngoscope —**

- 1 The divided airway is an angled pharyngeal airway split longitudinally into two parts held together by a pin. It is inserted into the mouth and through its lumen a rubber Magill tube is inserted blindly into the larynx. It can either be left in place or the pin can be removed so that the two parts will separate and can be withdrawn.
- 2 With the patient deeply anaesthetized and the anaesthetist standing to the left of the patient and facing him and an assistant drawing the tongue forwards the anaesthetist passes two fingers of his left hand over the dorsum of the tongue so that the epiglottis is hooked forwards. The tube which must be fully curved is guided by the two fingers in the mouth into the glottis. Curved introducers have been used to aid this procedure which was the one favoured by Fritz Kuhn.

Trup has suggested passing the tube blind through a London Hospital prop held in the mouth with the head of the anaesthetized patient in full extension. The tube must not be too fully curved.

**Technique of Orotracheal Intubation without Laryngoscope continued**

*During intubation*—about 70 per cent of patients show electrocardiographic changes mostly sinus tachycardia but occasionally premature ventricular systoles nodal rhythm and sinus bradycardia. Insufficient depth of anaesthesia trauma and hypoxia increase the incidence of these abnormalities.

**Extubation**—After the operation the tube can be removed in the theatre the air passages sucked clear and a pharyngeal airway inserted. Otherwise the patient returns to the ward with the tube still in position. In the latter case it is better that it should remain until the return of the cough reflex when it can be removed by the post operative observation ward sister.



Fig. 4.—Portex flexible metal endotracheal guard (British Oxygen Gases Ltd.)

Laryngeal spasm after extubation is sometimes seen so it is important to verify that breathing is free after the tube is removed. Extubation spasm can usually be prevented by the intravenous injection of 30–50 mg of suxamethonium. Inflation of the apnoeic patient with oxygen is then carried out until the return of normal breathing. This has the following advantages: (1) It prevents coughing and straining with increase of venous pressure strain on suture lines rise in intra ocular pressure rise in cerebrospinal fluid pressure. (2) It enables a careful toilet of the upper air passages to be carried out without disturbing the patient. (3) It minimizes post operative sore throat. On the other hand it is an unwise pharmacological venture to inject a depolarizing relaxant into a patient who has been for some time under the influence of a non depolarizing agent.

Difficulty in extubation has been reported in patients in whom intubation was easy. Acute oedematous laryngitis has resulted from this state of affairs.

**Some Difficulties in connexion with Intubation —**

- 1 Kinking of the tube causing respiratory obstruction. Use as firm a tube for orotracheal intubation as possible. A Portex tube kinks less easily than a rubber one. A pharyngeal pack may be used to support the tube. Kinking is rare with a naso tracheal tube. Bourne's flexible metal guard helps to prevent kinking (Fig. 4).
- 2 Respiratory obstruction caused by use of too small a tube.

- 3 Separation of angle piece from tube
- 4 Blockage of tube by blood mucus etc. Tube may need to be sucked out.
- 5 Obstruction has resulted from a tube being pulled up so that its distal end becomes blocked by the inflated cuff. For this reason the cuff should not be too near the distal end of the tube.
- 6 Slipping out of tube owing to weight of attached breathing tubes etc.
- 7 Slipping into nose or pharynx of tube. A safety pin or angle piece should prevent this. Occasionally forceps are needed to recover the tube.
- 8 Epistaxis. This looks messy but seldom interferes with the anaesthesia or causes post-operative discomfort.
- 9 Damage to teeth. If a tooth is knocked out it must be accounted for and prevented from disappearing into the trachea. If a radiograph shows it to be in a bronchus it should be looked for with a bronchoscope as soon as possible.
- 10 Partial occlusion of tube by nasal spurs deflected septa etc. causing respiratory obstruction. The opposite naris or the orotracheal route may have to be substituted.
- 11 Intubation of the right bronchus. Due to use of too long a tube. Average distance between central incisors and carina is  $10\frac{1}{2}$  in in an adult male and 9 in in a female. Distance from nares to carina is an additional  $1\frac{1}{2}$  in. In a newborn baby 2 in separates the gums from the cords and a similar distance separates the cords from the carina. Diagnosed by hypoxic appearance of patient, absence of air entry into left lung, unsatisfactory anaesthesia with jerky breathing.

**Maintenance of Anaesthesia**—With the tube in position the airway should take care of itself while laryngeal spasm loses its importance. Anaesthesia can be maintained—

- 1 By a continuous or intermittent flow gas machine via an angle piece and connecting tube or a face mask.
- 2 By the E.M.O. inhaler.
- 3 By the semi-open drop method on a mask placed over the tube.
- 4 By Flagg's can—a tin can with several openings in the top and containing ether. The endotracheal tube is connected to the can so that the patient breathes in air which is sucked over the surface of the ether.
- 5 By Ayre's T piece (see p. 215).

**Anæsthetic Agents in relation to Endotracheal Intubation—**

- 1 **SUXAMETHONIUM**—This short acting muscle relaxant is probably the most popular drug used for making intubation quick easy and atraumatic. If the chloride is used a suitable dose is 5–50 mg (i.e. 20–40 mg of active cation). If the bromide then the dose should also be 20–40 mg active cations (i.e. 30–60 mg of the salt). This is given after the patient is anaesthetized it works in less than one minute while its effect which may include apnoea seldom lasts more than a few minutes. For blind nasal intubation a combination of

*Anæsthetic Agents in relation to Endotracheal Intubation continued*

thiopentone and suxamethonium shows great possibilities. Dosage suggested is 0.25 g of the barbiturate with suxamethonium 12-16 mg of active cation. The relaxant lessens the likelihood of laryngeal spasm. The muscle pain which may cause distress the day following operation can be lessened if gallamine 10-20 mg is injected before the suxamethonium. An interval of 1 min should separate the injections. There are however some pharmacological objections to this sequence.

- 2 **CURARE THIOPENTONE**—This is probably the easiest and certainly the quickest method of direct vision intubation. A test dose of 6 mg of *d* tubocurarine chloride is given (T. C. Gray) (or gallamine 20 mg) and if uncontrollable ptosis and dyspnoea do not appear a further 9 mg to 19 mg are injected (or gallamine 40-80 mg). This is followed by 2½ per cent thiopentone solution injected through the same needle in amounts varying from 0.25 g to 0.75 g. Oxygen should then be given for two or three minutes from a mask using positive pressure on the reservoir bag if necessary. Laryngoscopy can then usually be performed with facility in the well oxygenated patient. Occasionally additional doses of the drugs are necessary. This method is not for the beginner who may be faced with an apnoeic patient with cords which cannot be visualized.
- 3 **NON DEPOLARIZING RELAXANTS**—Intravenous injection of 10-25 mg of *d* tubocurarine chloride rapidly produces muscular relaxation which helps laryngoscopy. Gallamine (flaxedil) 40-80 mg is also very suitable. Anæsthesia is usually produced by cyclopropane or nitrous oxide.
- 4 **ETHER**—This agent produces muscular relaxation without respiratory depression so is very suitable for either method of intubation. It is the best and safest agent for the beginner.
- 5 **BROMETHOL**—It depresses respiration so blind intubation may be difficult. Laryngeal irritability is not increased. In the edentulous laryngoscopy may be possible with this agent alone.
- 6 **CARBON DIOXIDE**—A few breaths of this gas before intubation by deepening breathing and by increasing the abduction of the vocal cords is a helpful measure.
- 7 **CYCLOPROPANE**—Depresses respiration and makes blind intubation difficult but the early relaxation of the jaw and neck muscles facilitates laryngoscopy. Anæsthesia lightens rapidly so a speedy technique is often necessary. Suxamethonium aids intubation.
- 8 **INTRAVENOUS BARBITURATES**—Intubation should seldom be attempted under thiopentone alone. Laryngeal irritability is increased while muscular relaxation is poor with these agents given in reasonable dosage. Respiration is depressed. The first makes blind intubation the second makes laryngoscopy difficult. Topical laryngeal analgesia helps to prevent spasm and blind intubation may produce dangerous hypoxia without it.

Hunter\* describes the following technique. The larynx is sprayed under light thiopentone anaesthesia (0.15–0.3 g) with a topical analgesic so that any coughing which may result is not associated with apnoea or hypoxia. An oropharyngeal airway is used for this either the Waters or Philips model. After an interval a further dose of thiopentone is injected (0.1–0.4 g) and when the jaw is relaxed the laryngoscope is inserted and intubation carried out. Severe breath holding does not occur. In patients with a full set of teeth laryngoscopy under thiopentone is difficult. It is relatively easy in the edentulous.

9. **NITROUS OXIDE-OXYGEN**—This agent used alone is not suitable for the passage of endotracheal tubes except in expert hands. Adequate analgesia of the pharynx and larynx with heavy premedication makes it more suitable. Seldom should the laryngoscope be used with nitrous-oxide-oxygen alone. The addition of trilene to gas-oxygen makes blind intubation relatively easy but does not easily produce relaxation suitable for laryngoscopy.

10. **PETHIDINE AND GAS OXYGEN**—Pethidine has a selective depressant effect on the larynx. Ruben and Andreassent recommend the use of 1 per cent solution injected slowly intravenously at a rate of 60 mg per minute. After usual premedication 50–80 mg are injected followed by nitrous oxide-oxygen then gallamine 30–60 mg (or tubocurarine 5–10 mg). After a minute a further 50 mg of pethidine goes in and then laryngoscopy and intubation are carried out. Respiratory depression is usual but does not last long. Coughing is infrequent.

11. **PHENOTHIAZINE DERIVATIVES**—Laryngoscopy and intubation is often possible after intravenous injection of chlorpromazine, promethazine and pethidine without the addition of thiopentone or a relaxant.

12. **HALOTHANE**—This agent fairly rapidly relaxes the pharyngeal and laryngeal muscles and can be used without relaxants for intubation.

Voluntary hyperventilation, breathing pure oxygen for a few minutes before intubation minimizes blood gas alteration associated with this manoeuvre.

**Endotracheal Intubation in Infants and Children**—The muscular exertion required to move columns of gas along tubes together with their small tidal exchange makes ordinary endotracheal technique unsuitable for babies unless respiration is controlled.

Avr's method (1937) does away with valves and rubber bags and allows the circuit to be open to the outside air. One end of the cross piece of a metal T tube 1 cm in diameter is connected to an angle piece of an endotracheal tube by about 1 in. of rubber. Nitrous oxide, oxygen and if necessary a volatile vapour are insufflated by a small inlet tube at right angles to the main limb (the upright of the T). The other cross piece



Endotracheal Intubation in Infants and Children *continued*

has fixed to it rubber tubing—open to the air—which constitutes a small reservoir for the anæsthetic gases most of which would otherwise escape into the outside air. The internal diameter of the reservoir tube should be 1 cm so that each inch in length will have a capacity of about 2 ml\* (For adults a slightly larger tube with an internal diameter of 1.25 cm and a capacity of 3 ml per inch in length may be used). These measurements should not be exceeded otherwise increased

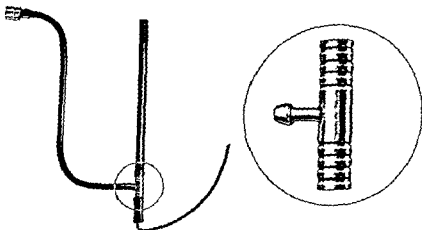


Fig. 43—Ayre's T-piece (Bentley Oxygen Gases Ltd.)

dead space will result. The fresh gas inflow should average twice the minute volume of the patient. Ayre gives the following table\* as a guide to the gas inflow and tube (reservoir) capacity required for children of different ages.

Age	Gas Inflow in litres/min	Reservoir Tube Capacity in ml
0-3 months	3-4	6-8
3-6	4-5	12-18
6-12	5-6	18-24
1-2 years	6-7	4-42
2-4	7-8	42-60
4-8	8-9	60-72

The upright of the T piece is connected to a continuous flow gas machine (Fig. 43). After induction of anaesthesia the endotracheal tube is passed and connected up. Nitrous oxide-oxygen in varying proportions—about 4 to 6 litres per minute—is allowed to bubble through later to pass over ether.

Ayre mentions the following advantages —

- 1 No obstruction to respiration or hypoxia
- 2 Amount of re-breathing adjustable by length of breathing tube
- 3 Vascular congestion reduced

The technique can be used in adults and is especially useful in intra cranial surgery. The patient should be carried to plane II stage 3 before being connected to the tube.

In children the laryngeal reflexes are very active while the glottis is smaller relative to body size. Thus a tube may pass readily into a nares but be too big for the larynx. The largest tube in a child's larynx because of its rubber walls reduces the capacity of the larynx very considerably.

The larynx is placed far forwards in infants and children.

Gillespie advises the following sizes of tubes for the following ages —

Less than 3 months	Size OO
From 3 to 9	OA
9 15	O
15 21	1
1½ 2½ years	2
2½ 4	3
4 6	4
7 9	5
10 12	6-7
13 14	8

A length of 12 cm. is sufficient for a Size OO tube.

**Choice between Nasotracheal and Orotracheal Intubation —**

Nasotracheal intubation is likely to be less traumatic than oro tracheal intubation in regard to lips and teeth more traumatic to pharynx and nose. It should be avoided when there is infection in the nose or sinuses or nasal deformity.

Orotracheal intubation may allow a larger tube to be passed and is the method of choice when a cuffed tube is to be used. Extra care and skill are needed in patients with short beefy necks and those with teeth easily traumatized.

The route likely to be easier should be used. The method most convenient to the surgeon should be used unless it is contra indicated on other grounds.

**Advantages and Disadvantages of Intubation.—**

**ADVANTAGES —**

- 1 Avoidance of respiratory obstruction and consequent absence of laboured respiration and capillary oozing
- 2 Absence of straining due to laryngeal spasm
- 3 Artificial respiration and control of intrapulmonary pressure made easy. Inflation of the stomach can easily be prevented
- 4 Enables anaesthetist to keep away from the operative field
- 5 Lessens the chance of inspiration of foreign matter and enables bronchial tree to be kept clear of blood or mucus by suction
- 6 Dead space reduced to a minimum (when face mask is not used)
- 7 Enables obstruction of tracheobronchial tree to be overcome as in obstructive goitre

*Advantages and Disadvantages of Intubation continued***DISADVANTAGES —**

- 1 Trauma to lip nose throat and larynx resulting in hoarseness dysphagia pain etc Granulomata of the cords (granuloma pyogenicum) are said to have resulted from the trauma of intubation Hoarseness may be delayed for four months after intubation Prognosis is good if adequate removal is undertaken Abrasion of the mucosa of the pharynx may result in extensive surgical emphysema This can be partially relieved by aspirating air from beneath the deep fascia of the neck with a syringe and wide bore needle A subglottic slough may follow a cuffed tube if the cuff is too tightly inflated
- 2 Safe use of laryngoscope may require a deeper plane of anaesthesia than the surgical operation
- 3 Lack of contact of inspired gases with the mucosa covering the turbinates leads to cold dry gases reaching the alveoli the use of a closed circuit prevents this

There is no definite evidence that intubation increases the post operative pulmonary morbidity

**Reflex Circulatory Responses to Laryngoscopy and Intubation —**

During light general anaesthesia and also under local analgesia direct laryngoscopy and intubation uncomplicated by hypoxia hypercapnia or cough is capable of causing a rise in blood pressure and an increase in heart rate and arrhythmia because of stimulation of the nerve endings of the vagus These changes are not of great clinical significance and are made much worse by changes in the blood gases For this reason inflation with oxygen should be carried out following the injection of a relaxant and before intubation Deep anaesthesia reduces the incidence of these reflexes and should be used in certain cases of myocardial disease for this reason \* No vagal inhibition occurs Arrhythmias follow intubation not laryngoscopy Sudden death presumably from ventricular fibrillation has been reported to result reflexly from intubation and from endotracheal suction

Electrocardiographic studies during intubation reveal that about 60 per cent of patients show some disturbance of a transitory nature Most frequent are sinus tachycardia premature ventricular contraction nodal rhythm and sinus bradycardia Light planes of anaesthesia clumsy technique and hypoxia make the condition worse Preliminary intravenous injection of 1 per cent procaine reduces their incidence while cocaineization makes them worse † Cyclaine (hexylcaine hydrochloride) used as a topical analgesic is said to reduce the incidence of arrhythmia during intubation Procaine amide 300 mg (in 10 per cent solution) injected intravenously one to five minutes before intubation reduces the incidence of arrhythmias

Extubation at light planes of anaesthesia causes no significant ECG changes and so is safe

**Laryngeal Complications of Intubation —**

1. **NON SPECIFIC GRANULOMA OF THE LARYNX (GRANULOMA PYOCENICUM)**—This is usually superimposed on a contact ulcer and takes some time to develop. The usual site is the tip of the vocal process of one or both arytenoids in the posterior one third of the rima glottidis. The tips of the vocal processes are prominent and are covered with mucoperichondrium. They are normally projected forcibly into the laryngeal lumen and slapped together. Contact ulcer is not necessarily due to trauma from the intubation but may be due to actual movements of the cords against the tube as it lies in the larynx perhaps twenty times a minute for two or three hours. These movements are not necessarily abolished by anaesthesia.\*

The prognosis of a contact ulcer is good. Healing is usual but is hindered by phonation. If it is followed by granuloma formation local removal will be necessary.

2. **ACUTE OEDEMATOUS STENOSIS**—More usual in children because of
  - (1) The amount of loose areolar tissue in the subglottic region
  - (2) The small lumen which is easily occluded. Obstructive laryngeal oedema from whatever cause has the following signs
    - a. Indrawing during inspiration at any of the following sites
      - (i) The suprasternal notch
      - (ii) Around the clavicles
      - (iii) The epigastrium
      - (iv) The intercostal spaces
    - b. Ashy grey pallor
    - c. Choking and waking in terror every time the child falls asleep
    - d. Restlessness

**TREATMENT**—No sedatives. Further intubation contra indicated. Low tracheotomy without delay.

**Indications for Endotracheal Anaesthesia —**

1. In operations—all but the shortest—in which a free airway cannot be otherwise maintained. Increasing skill lessens this indication.
2. In many abdominal operations to ensure quiet breathing and absence of straining.
3. In cases of intestinal obstruction with regurgitant vomiting performed under general anaesthesia intubation and occlusion of the larynx prevent aspiration of infected material. In such cases it may be wise to intubate under topical analgesia inducing general anaesthesia through the tube. This prevents vomiting during induction before the tube is in place.
4. In thoracic operations so that the airway is always patent suction can be easily carried out and control of intrapulmonary pressure is made easy.
5. In cases operated on in positions making control of the airway difficult e.g. operation in prone position.
6. When controlled breathing is to be employed. To hold the jaw and support the airway while rhythmically pressing on the breathing bag is uncomfortable and may be dangerous.

Indications for Endotracheal Anæsthesia *continued*

- 7 In operations on the head and neck
  - a Intranasal operations airway is secured and packing prevents aspiration of blood
  - b Tonsil dissection in adults In children undergoing dissection surgeons vary in their prejudices
  - c Oesophagoscopy and gastroscopy when not performed under local analgesia.
  - d Major dental operations
  - e All intracranial operations
  - f Radical mastoidectomy the tube should avoid the nostril of the diseased side if inserted through the nose
  - g Certain eye operations e.g. for squint dacryocystitis removal of the eyeball
  - h Thyroid surgery There are two schools of thought one recommending intubation because of the good airway and position of the anaesthetist remote from the site of operation the other avoiding intubation because of its tendency to cause post-operative tracheitis and dysphagia Pressure on the trachea by the tumour is an indication for intubation
- 8 In patients likely to develop laryngeal spasm e.g. some cases of cystoscopy hæmorrhoidectomy etc

Endotracheal anæsthesia should not be abused and should not be employed without a real indication

## INTUBATION IN NON SURGICAL CONDITIONS —

- 1 In grave asphyxia neonatorum The size 00 Magill tube with a knitting needle used as a stylet
- 2 In resuscitating patients in whom respiration is obstructed depressed or absent and who need frequent aspiration
- 3 In grave laryngeal obstruction due to inflammatory exudates tubes have been left in the trachea for four days and even longer without serious results
- 4 In patients with atelectasis and signs of exudate in lungs A nasotracheal tube passed blind after topical laryngeal analgesia will serve as a channel down which a suction catheter can be passed This will sometimes make unnecessary the more difficult bronchoscopic suction
- 5 For bronchography in children for the instillation of radio opaque oil—this is one of many techniques

Where an artificial airway is likely to be required for longer than twenty four hours a tracheostomy is often preferable to prolonged intubation

**Contra Indications —**

- 1 Aneurysm of aortic arch trauma of tracheal walls may cause rupture
- 2 Acute laryngitis trauma may make condition worse or cause cedema
- 3 In cases of open pulmonary tuberculosis trauma may lead to tuberculous laryngitis

## CHAPTER VII

## RECTAL ANÆSTHESIA—BASAL NARCOSIS

**History**—Pirogoff gave ether vapour into the rectum in 1847 Wanscher of Copenhagen revived it in 1882 and Mollière of Lyons used it again in 1884 Gwathmey introduced rectal oil-ether in 1913 Butzrenger and Eicholtz (head of pharmacological section of Bayer Products at Wuppertal Elberfeld) used avertin in 1927 Rowbotham used paraldehyde in oil in 1928 Shipway and Blomfield used avertin in England in 1928  
 Basal narcosis is deep premedication pushed to the stage of unconsciousness

## ANATOMY OF RECTUM

The rectum is a hollow muscular tube 5 in long extending from the third sacral vertebra where it is continuous with the sigmoid and ending below where it bends sharply backwards into the anal canal. It lies in the curve of the sacrum and coccyx and is S shaped. Its lower end is dilated to form the rectal ampulla. The average rectum holds about 6 fl oz.

When empty the rectal mucosa is gathered into longitudinal folds. There are also three transverse folds the valves of Houston which overlap when the viscus is empty and help to support the faecal mass when it is full.

**Relations.—**

To the peritoneum —

- a The upper third is covered by peritoneum in front and at the sides
- b The middle third is covered only anteriorly and from it the peritoneum is reflected on to the seminal vesicles in the male and the posterior vaginal wall in the female
- c The lower third has no peritoneal covering

Posterior relations are —

- a The superior hæmorrhoidal vessels
- b The left pyriformis muscle and left sacral plexus
- c The coccyx and sacrum
- d The levatores ani

Anterior relations are —

- a The rectovesical fold in the male and below it the seminal vesicles bladder and prostate
- b The pouch of Douglas in the female and below this the posterior vaginal wall. The pouch of Douglas may contain coils of intestine

**Supports**—The rectum is held in position by —

- 1 The attachments of the levatores ani
- 2 A layer of the endopelvic fascia

*Anatomy of Rectum continued*

- 3 The recto urethralis muscles
- 4 A fibrous cord on each side of the rectum running from the third sacral vertebra to the rectum just above the levator. Through this fibrous tissue run the nervi erigentes (S 2-3) and the middle hæmorrhoidal arteries
- 5 The connective and fatty tissue of the pelvis

**The Anal Canal**—Runs from the apex of the prostate to the anus. Is about 1 in long and runs sharply backwards and downwards from its junction with the rectum. It is surrounded from above downwards by the internal sphincter, the levatores ani and the external sphincter. Posteriorly is the ano coccygeal body, anteriorly the urethral bulb and membranous urethra in the male, the perineal body in the female.

The upper part is lined by mucosa which is thrown into vertical folds, the rectal columns of Morgagni; these end in valve like reflections, the anal valves.

**Blood supply**—The superior hæmorrhoidal artery, branch of the inferior mesenteric, supplies the rectum.

The middle hæmorrhoidal artery from the internal iliac and the inferior hæmorrhoidal from the internal pudic supply the anal canal.

The veins drain the hæmorrhoidal plexus, which is one of the communications between the portal and systemic circulations.

The hæmorrhoidal plexus consists of two parts, one internal to the muscular layer, the other external to it. The internal part drains into the superior hæmorrhoidal vein which goes to the portal system via the inferior mesenteric vein. The external part drains into the superior, middle and inferior hæmorrhoidal veins, which follow the course of the corresponding arteries.

**Nerve supply**—

The intrinsic nerve supply is—

- 1 Auerbach's plexus in the muscular layer
- 2 Meissner's plexus in the submucosa

These plexuses can take over automatic control of evacuation if impulses from the cord are removed.

The extrinsic nerve supply is—

- 1 Motor. From the pelvic nerves or nervi erigentes (S 2-3)
- 2 Inhibitory. From the second and third lumbar via lumbar splanchnic to the inferior mesenteric ganglion and thence to bowel via the inferior mesenteric and presacral (hypogastric) nerve. Paralysis of these fibres in a spinal analgesia may cause defæcation.

- 3 Afferent impulses from rectum travel via the nervi erigentes (S 2-3)

Afferent impulses from the anus and anal canal travel via the pudendal nerve and enter the cord with the S 2, 3 and 4 roots.

- 4 The internal sphincter receives motor fibres from the sympathetic via the inferior mesenteric and presacral nerves. The nervi erigentes are inhibitory.

- 5 The external sphincter is composed of striated muscle and is partly under voluntary control. It is supplied by the inferior hæmorrhoidal branch of the pudendal (S 2 3-4) which also supplies the skin round the anus

#### Lymphatics —

- 1 From the anus to the superficial inguinal lymph glands
- 2 From the anal canal to the hypogastric lymph glands (internal iliac glands)
- 3 From the rectum to the pararectal glands glands in the sigmoid mesocolon and thence to the glands around the origin of the inferior mesenteric artery

### ✓ BROMETHOL

(Aterlin—Tribromethanol)

*Butzen*

This is tribromomethyl alcohol  $\text{CBr}_3\text{CH}_2\text{OH}$ . It was synthesized by Willstaetter and Duisberg in 1923 and was first used by Butzengeiger and Eicholtz in 1922, as a complete anæsthetic. It is now used for basal narcosis and requires in addition the use of a supplementary anæsthetic. Its popularity is dwindling and rectal and intravenous thiobarbiturates are taking its place.

**Preparation** — Tribromacetaldehyde is reduced with the aid of aluminium ethoxide in absolute ethyl alcohol in a nitrogen atmosphere; the reaction product is then treated with aqueous sulphuric acid and the drug separated (Adriani). It is a white crystalline powder sold dissolved in amylene hydrate so that 1 ml of the solution contains 1 g of bromethol. The solution may decompose if exposed to light and heat or if left to stand when dissolved in water. Amylene hydrate  $(\text{CH}_3)_2\text{C}(\text{OH})\text{C}_2\text{H}_5$  is itself an anæsthetic and was reported on by John Snow in 1856. In the solution used for basal narcosis it does not greatly influence the anæsthetic potency nor does it interfere with the pH indicator used. It is volatile and may evaporate if kept exposed to air. Supplied in 100 ml containers together with 10 ml of Congo red solution. Should not be exposed to light.

**Dosage and Administration** — The dosage is 80–120 mg per kilo body weight (0.6 g per st) depending on the metabolic rate of the patient. Elderly feeble and obese subjects require a small dose. Feverish patients and those in pain require a higher dose. Children tolerate it well and need full doses. Morphine if given in addition must be prescribed in small quantities to prevent respiratory depression.

It is given in 2½ per cent solution in distilled water which must be at 40°C and not allowed to cool. Can be safely given in a 3 per cent solution which is less bulky. Vigorous shaking is necessary to dissolve all the droplets. A higher temperature may cause decomposition; a lower one may fail to allow complete solution. Decomposition products are dibromacetic aldehyde and hydrobromic acid, the former being irritating to the rectal mucosa. To prevent this irritation 2 drops of Congo red (1–1000 solution) are added to 5 ml of the prepared solution just before instillation. The colour should be a clear bright red.



*Anatomy of Rectum continued*

- 3 The recto urethralis muscles
- 4 A fibrous cord on each side of the rectum running from the third sacral vertebra to the rectum just above the levator Through this fibrous tissue run the nervi erigentes (S 2-3) and the middle hæmorrhoidal arteries
- 5 The connective and fatty tissue of the pelvis

**The Anal Canal**—Runs from the apex of the prostate to the anus Is about 1 in long and runs sharply backwards and downwards from its junction with the rectum It is surrounded from above downwards by the internal sphincter the levatores ani and the external sphincter Posteriorly is the ano-coccygeal body anteriorly the urethral bulb and membranous urethra in the male the perineal body in the female

The upper part is lined by mucosa which is thrown into vertical folds the rectal columns of Morgagni these end in valve-like reflections the anal valves

**Blood supply**—The superior hæmorrhoidal artery branch of the inferior mesenteric supplies the rectum

The middle hæmorrhoidal artery from the internal iliac and the inferior hæmorrhoidal from the internal pudic supply the anal canal

The veins drain the hæmorrhoidal plexus which is one of the communications between the portal and systemic circulations

The hæmorrhoidal plexus consists of two parts one internal to the muscular layer the other external to it The internal part drains into the superior hæmorrhoidal vein which goes to the portal system via the inferior mesenteric vein The external part drains into the superior middle and inferior hæmorrhoidal veins which follow the course of the corresponding arteries

**Nerve supply —**

The intrinsic nerve supply is —

- 1 Auerbach's plexus in the muscular layer
- 2 Meissner's plexus in the submucosa

These plexuses can take over automatic control of evacuation if impulses from the cord are removed

The extrinsic nerve supply is —

- 1 Motor From the pelvic nerves or nervi erigentes (S 2-3)
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- 2 Support the circulation —
  - a Pressor amines
  - b Intravenous saline or plasma
  - c Tilt patient head down
- 3 Rectal lavage with warm hypertonic sodium thiosulphate solution

#### Advantages —

- 1 Removes anxiety of induction of anaesthesia
- 2 Produces post-operative sedation and amnesia
- 3 Unlike paraldehyde has no unpleasant odour
- 4 Helps to produce muscular relaxation

#### Disadvantages —

- 1 Expert nursing required before and after operation
- 2 Danger of liver and kidney damage
- 3 Excretion cannot be hurried as it is non volatile
- 4 Post-operative pulmonary morbidity increased especially in abdominal cases

#### Indications —

- 1 Thyrotoxic cases Modern pre operative preparation of these patients is making it less useful than was formerly the case
- 2 Frightened anxious patients with an abnormal dread of anaesthesia
- 3 Short intracranial operations bleeding is reduced e.g. fenestrations
- 4 Ophthalmic operations because intra ocular tension is decreased and the vessels of the conjunctiva are contracted and so bleed minimally

#### THERAPEUTIC USES OF BROMETHOL —

- 1 To control the contractions of tetanus strychnine poisoning eclampsia and status epilepticus
- 2 To release the muscles of the bronchioles in status asthmaticus
- 3 To quell delirium in acute alcoholism and toxic states etc

#### Contra indications —

- 1 Low basal metabolic rate because of delay in excretion
- 2 Liver and kidney disease
- 3 When a quick return of reflexes is required after operation e.g. upper abdominal cases throat and nose operations
- 4 Some anal and rectal diseases
- 5 When adrenaline is to be used lest the combination produce ventricular fibrillation

In obstetrics bromethol must be used in small dosage lest it should depress the respiration of the child It may produce restlessness if pain is left uncontrolled

#### PARALDEHYDE

Paraldehyde was discovered by Wiedenbusch in 1829

Rowbotham was the first in 1928 to use paraldehyde in oil per rectum to produce basal narcosis Harold Sington used it for children in the following year while in 1932 Rosenfield and Davidoff gave it per rectum to obstetrical patients

The formula is  $(CH_3CHO)_3$  and it is prepared by the action of concentrated sulphuric acid on acetaldehyde which causes the polymerization

**Bromethol continued**

blue colour shows acid formation and the solution must be discarded. The warmed solution can be stored for 12 hours in a thermos flask but must be tested just before use.

Bromethol in 1 per cent solution in saline can be given intravenously as a continuous drip.

The evening before administration an enema is given. The solution is run into the rectum from a funnel through a catheter the patient being on his left side with the foot of the bed raised. It is run in slowly to prevent too rapid absorption. It is a depressant of the central nervous system. It is rapidly absorbed from the colon 50 per cent of it disappearing from the bowel during the first 10 minutes after injection.

The patient falls asleep in 10–15 minutes without any stage of excitement. The maximum effect is shown in 30 minutes and at the end of an hour its effects begin to wear off as it is detoxicated. The patient will usually be awake three hours after administration but may go to sleep again. It gives good amnesia. Large doses give full surgical anaesthesia but it is accompanied by profound respiratory depression. Normal narcosis with bromethol occurs when blood concentration is 6–9 mg per 100 ml. Constant supervision by a skilled person is required to prevent *respiratory obstruction consequent on the muscular atony*.

**Pharmacology —**

**CIRCULATION**—The blood pressure falls during the first half hour but gradually picks up again. The fall is most severe in hypertensive subjects. It is capable of being controlled by pressor amines and is due to depression of the vasomotor centre in the medulla and relaxation of the muscular walls of the smaller blood vessels. In addition the cerebrospinal fluid pressure is increased.\*

**RESPIRATION**—Breathing depressed in overdosage respiratory paralysis results. This is the great danger of the drug. Relaxation of the jaw may result in respiratory obstruction so that careful nursing attention is necessary to see that hypoxia is not present.

**METABOLISM**—Body temperature is reduced, the blood sugar is slightly raised, the basal metabolic rate is lowered.

The drug is toxic to the diseased liver and to the normal liver in excessive dosage it inhibits the secretion of bile acids. *Ketosis may follow administration. Rarely liver damage has been reported even in patients with no apparent liver disease. In the liver bromethol unites with glycuronic acid which is excreted by the kidneys. Kidney disease retards excretion. The amylene hydrate is excreted by the lungs and kidneys in an unchanged condition. It causes muscular atony.*

**Treatment of Overdosage —**

1. Assist respiration and prevent hypoxia. Endotracheal intubation if required.

of dosage the strong active muscular hyperthyroid person requiring more than the obese feeble or anæmic patient. Contra indicated in severe cardiac disease severe respiratory embarrassment and grave anæmia

#### SPECIAL INDICATIONS —

- 1 For cardiac catheterization in young children
- Before eye examinations in young children
- 3 To produce quietness in young children during X ray examination
- 4 For cystoscopies
- 5 For encephalography
- In combination with cyclopropane anæsthesia it gives moderately good relaxation in abdominal surgery
- 6 Has been given in one half to two thirds the dosage used in surgery for vaginal delivery in obstetrics where it appears to have no bad effect on the baby
- 7 Before dissection of tonsils in children (provided the post operative nursing is good)

#### ETHER OIL

Rectal instillation of ether vapour was advocated by Pirogoff of St Petersburg in 1847 and of ether and water by Dupuy of Paris in the same year. It was revived by Gwathmey in 1913.

Ether 65 per cent is mixed well with olive oil 35 per cent and 20 c.c. of the mixture per stone of body weight is the average dose. Less should be given to obese and feeble patients. Anæsthesia which may be complete lasts for 2½–3 hours. A 50–50 mixture with olive oil can also be used.

The skin of the buttocks should be smeared with Vaseline and the mixture run in from a funnel and catheter. As the solution enters the rectum cramp may be experienced.

It is used in obstetrics in status asthmaticus to induce anæsthesia in patients with cardiac decompensation.

#### CHAPTER VIII

### INTRAVENOUS ANÆSTHESIA

**History** —Ore Professor of Physiology at Bordeaux used chloral hydrate intravenously in 1872 in a patient suffering from tetanus the hypodermic syringe and needle having been invented by Pravaz and Alexander Wood respectively in 1853.

Intravenous hedonal was used in 1905 by Krawkow of St Petersburg, Russia and in 1912 by Max Lake in London. In 1909 Burckhardt gave chloroform and ether by the intravenous route.

The first barbiturate was synthesized by Emil Fischer and v. Mering in 1903. This was diethyl barbituric acid or veronal. Pheno-barbitone was discovered in 1911. Somnifaine was the first

*Paraldehyde continued*

of three molecules of acetaldehyde into one of paraldehyde. It was introduced as a sedative into medicine by Cervello in 1882.

Paraldehyde is a clear volatile inflammable liquid having a characteristic odour. It should be stored away from heat, light and air. A relatively fresh preparation should be used as old paraldehyde may decompose with the formation of acetic acid which may cause rectal sloughing.

It is administered like bromethol and is dissolved in either saline or olive oil usually 1 drachm of paraldehyde to 1½ oz of solvent. The average dose is 50–60 minims for each stone of body weight 8 drachms being the maximum. It should be given three-quarters of an hour before operation.

It has a low vapour pressure at room temperature and so cannot be used as an inhalation anæsthetic. For the same reason although it burns it will not explode.

It acts more slowly and for a longer time than avertin and is only slightly depressing to respiration. It has a constricting effect on the bronchi. Muscle tone is not lost and reflexes are not abolished. It is excreted unchanged by the lungs and also by the liver.

It is a very useful and safe basal narcotic for children its disadvantages being its smell, the long period of post-operative sedation and the large volume of fluid to be instilled into the rectum. Is used during labour. It can be given in doses of 5–10 ml either intravenously (diluted 1–10 in saline) or intramuscularly together with a little hyaluronidase to speed up its action if necessary and is a good anti-convulsant.

### **THIOPENTONE (PENTOTHAL) AND EVIPAN (HEXOBARBITONE)**

Rectal use first described by Weinstein and Adams in 1939.

Most useful in basal narcosis with rapid onset of sleep and absence of prolonged post-operative sedation. Rectal thiopentone can be used to produce either pre-anæsthetic hypnosis in doses of 13.3 mg per lb or 1 g per 75 lb or basal anæsthesia in doses of 20 mg per lb or 1 g per 50 lb of body weight. With the first the patient is sleeping but is rousable with the second he cannot easily be roused but will react to painful stimuli. Skilful nursing supervision is necessary after the drug is given. Can also be given in suppository form\*.

**Dosage**—Dissolve 1 g in 50 (or 75) ml of tap water and give 1 ml for each 1 lb of child. More concentrated solutions may be used. It should be instilled into the rectum 15–30 min before operation. The small bulk is an advantage in children the strength of solution being anything between 1.5 per cent and 10 per cent. In children maximum rectal dose should be 1.5 g in adults 3–4 g. It should be preceded by atropine and given on an empty stomach no preliminary enema being necessary. It causes less restlessness than pentobarbitone because it is excreted quicker. Its effects last one hour and a child will usually take food in three hours although remaining drowsy for the rest of the day. Judgement is required in assessment

of dosage the strong active muscular hyperthyroid person requiring more than the obese feeble or anæmic patient. Contra indicated in severe cardiac disease severe respiratory embarrassment and grave anæmia

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*History continued*

barbiturate to be used intravenously it is a combination of diethyl and diallyl barbituric acids and was used by Daniel and G Bardet in 1921

In 1927 Bumm introduced pernocton while Zerfas in the United States used sodium amytal intravenously two years later This was soon followed by the use of nembutal (pentobarbitone) (1930) by Volwiler and Tabern

Hirschner gave avertin (bromethol) intravenously in 1929

Evipan was the first drug to make intravenous anæsthesia popular and was used by Weese and Scharpf in 1932 having been synthesized by Kropp and Taub It was first used in Great Britain by R Jarman and L Abel (1933)

Pentothal odium (thiopentone) was synthesized in 1932 by Volwiler and Tabern and introduced into clinical practice by Lundy of the Mayo Clinic and by Waters of Madison in 1934 First used in Great Britain by Jarman and Abel in 1935

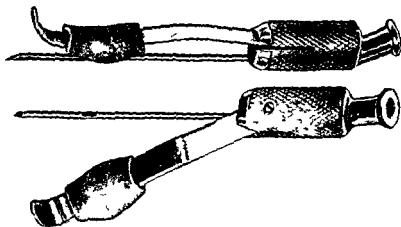


Fig 44 — Mitchell intravenous needle

**Apparatus** — Syringes of 10 ml and 50-ml capacity with eccentric nozzles Large bore needle for preparing solution File Intravenous needles with short bevel  $1\frac{1}{4}$  in long Sterile swabs and antiseptic lotion Arm board or arm table and suitable pad or folded towels to prevent outstretched arm being strained backwards Rubber tubing and artery forceps to use as tourniquet Dental prop or Baker's airway Mouth gag Pharyngeal airways and tongue forceps Facilities for the insufflation of oxygen into the lungs The Mitchell (Fig 44) or Lundy needle (Fig 45) or one of its modifications is useful if intermittent intravenous anæsthesia is contemplated so that clotting in the intravenous needle is prevented All intravenous apparatus should be autoclaved

To give serial injections of thiopentone relaxant etc without having the arm abducted a needle placed into a suitable vein in the arm can be connected by a length of polythene tubing to a syringe and three way tap on the anesthetic trolley (Lee) • (Fig 46)

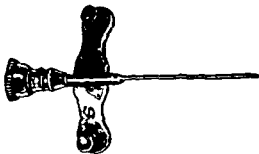


Fig 45—Gordh's intravenous needle for continuous injections (British Oxygen Gases Ltd)

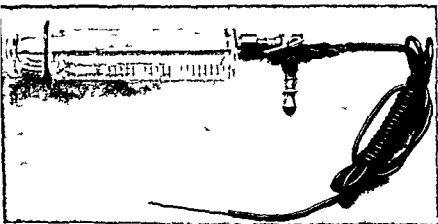


Fig 46—The Lee television apparatus for intravenous injections with the patient's arm at his side

**Anatomy and Physiology**—The great majority of injections are made into the superficial veins of the forearm. These are anterior to the deep fascia and usually include (1) The cephalic on the radial side (2) The basilic on the ulnar side (3) The median cubital (basilic) vein (4) The median antebrachial vein.

The *cephalic* vein drains the radial side of the dorsal venous network of the wrist and hand and passes up the radial side of the forearm anterior to the elbow joint where it lies between the brachioradialis and the biceps brachii. It gives off the median cubital



*Anatomy and Physiology continued*

vein (median basilic vein) in front of the elbow joint and this passes medially to join the basilic vein. Passing up the arm the cephalic vein enters the axillary vein below the clavicle after piercing the coracoclavicular fascia.

The *basilic vein* drains the ulnar side of the dorsal venous network and running up the dorsal aspect of the forearm at first becomes anterior below the elbow joint. It is joined by the *median cubital vein* and passes between the pronator teres and the biceps brachii piercing the deep fascia in the middle of the arm. It then ascends medial to the brachial artery and at the lower border of the teres major becomes the axillary vein.

The *median antebrachial vein* drains the blood from the volar aspect of the wrist and hand. It ascends in front of the forearm and joins either the cephalic or basilic vein.

The arrangement of these veins is often irregular.

The following factors cause the blood in the veins to move proximally —

- 1 Pressure transmitted from the arterial system via the capillaries
- 2 Massaging effect of muscles
- 3 Valves preventing backflow
- 4 Negative intrathoracic pressure during inspiration
- 5 Negative intra auricular pressure during diastole

Very rapid intravenous injection of fluid may cause shock.

**Technique of Intravenous Injection** — The easiest and most accessible vein should be chosen and will usually be found anterior to the elbow joint. Veins in this situation should only be used if a clean and easy venepuncture is made. To burrow deeply and blindly into the tissues in this area is to run a danger of injury to the median nerve or an artery. Other possibilities are —

- 1 The cephalic vein on the radial side of the forearm
- 2 The internal saphenous vein anterior to the internal malleolus. The skin overlying this is tough so a specially sharp needle must be used.
- 3 Veins on the back of the hand or front of wrist
- 4 The external jugular vein

There is usually retrograde amnesia for venepuncture.

**Technique for keeping vein open** (1) Drip with a three way tap  
(2) Mitchell needle (3) C ordh needle

When the arm is chosen it should be resting firmly on an arm table or arm board so padded that the elbow can be hyperextended. The veins are made as prominent as possible by the use of a venous tourniquet such as a length of rubber tubing twisted round the arm. If the anaesthetist is single handed it can be secured and released by means of an artery forceps. Applied near the site of injection the tourniquet steadies the vein proximally while the anaesthetist's finger by stretching the skin steadies it distally. This is very important in thin old people whose veins although prominent very readily slip about beneath the skin and may run away from the point of the needle.

## Aids to visualization of veins —

- 1 Tourniquet—see that artery is not occluded also as the presence of arterial pulsation is a most useful guide as to the position of an artery and hence to avoidance of injection into it
- 2 Massaging of forearm in proximal direction and gentle slapping of tissues to promote vasodilatation
- 3 Clenching of fist
- 4 Holding arm downwards before application of tourniquet
- 5 Application of massive hot fomentation to whole forearm for half an hour before injection
- 6 Use of radiant heat before injection

Any form of general anaesthesia will cause dilatation of the superficial veins and so will make venepuncture easier

When the skin has been well cleaned with an antiseptic the needle is inserted so that it comes to lie between the skin and the vein wall needle bevel can be either towards the skin or away from it For this the syringe should be lying almost parallel to the skin unless the vein is deeply placed The point is now advanced and the vein wall is pierced at a different level from the skin puncture This tends to prevent transfixion of the vein and lessens haematoma formation when the needle is withdrawn With the needle point within the lumen of the vein it should if possible be advanced for a short distance to prevent slipping out

If more than a single dose of drug is to be given the syringe is now fixed to the arm This is most easily done by securing not too tightly the rubber tourniquet round the syringe arm and arm table with an artery forceps A similar device steadies the wrist A small vein requires a small needle while clotting is prevented by frequent injection of small volumes of solution or by fitting a through and through tap between syringe and needle which is only open during injection so preventing blood from entering the needle and clotting there If needle does become blocked it can often be freed by injecting a small volume of saline into it from a 1 ml or 2 ml syringe—not one of larger size A large bore needle e.g. 20 s.w.g. is less likely to clot than a small one One minim of procaine can be injected with an intradermal needle before the larger needle is inserted if necessary With the needle in the vein the aspiration test is performed and injection can commence After withdrawal of the needle oozing should be checked by firm pressure and elevation of the limb rather than by acute flexion of the elbow

**THIOPENTONE SODIUM\***

*(Thiopental Trapanal Penthiobarbital Intraval Nesdonal Farmolal)*

This is sodium ethyl (1 methyl butyl) thio barbiturate It is the sulphur analogue of nembutal It is 30–50 per cent more potent than hexobarbitone (evipan) and holds the field at present as the best ultra short acting intravenous barbiturate Introduced commercially as pentothal sodium in 1934

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**Anatomy and Physiology continued**

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**Thiopentone Sodium** *continued*

It is a yellow amorphous powder with odour resembling  $H_2S$ . It is soluble in water and alcohol and forms a 2½ per cent solution in distilled water of pH 10.4–10.6 (pH of blood 7.4). A 2.8 per cent solution in water is isotonic. To prevent formation of free acid by carbon dioxide from the atmosphere 6 per cent sodium carbonate is added to the powder which is prepared in an atmosphere of nitrogen. In solution it is not very stable but can be left for 24–48 hours or longer without harm resulting on subsequent injection provided solution remains clear. The oil/water coefficient is 4.7 (of barbitone it is 0.214).

It is supplied in ampoules of 0.5, 1.0 and 5.0 g. with sterile distilled water sufficient to make a 5 per cent solution or a 2.5 per cent solution. In the U.S. a 2.5 per cent solution is more commonly used and is in the writer's opinion to be preferred. A solution which is cloudy e.g. after standing for three days at room temperature or seven days in a refrigerator—should be discarded.

To carry out a safe smooth anæsthesia with this agent may prove to be more difficult than it looks. Anæsthesia with thiopentone should never be undertaken lightly.

**Pharmacology**—Like other barbiturates it causes sedation, hypnosis, analgesia, anæsthesia and respiratory depression depending on the dose injected and the rate of injection. Cerebral oxygen consumption is lessened, even when the arterial oxygen tension is greater than normal. This is due to interference with cerebral cellular oxygen utilization. Whereas the volatile anæsthetics act chiefly on the cerebral cortex, the barbiturates are thought to act chiefly on the hypothalamus. After a single small dose of thiopentone its level in the plasma falls rapidly and the patient regains consciousness. This fall is due to the concentration of the drug in the body fat where it may be six to twelve times that in the plasma. Thus the ultra-short action is due not to rapid destruction in the tissues but to rapid concentration in the body fats. The plasma level of the drug after diffusion equilibrium between the tissues and the fat is established is less than that required to produce anæsthesia and the patient wakes up. After a single large dose or repeated small ones however the resulting equilibrium plasma level is high enough to cause anæsthesia and is so maintained from the reservoir of drug in the fat and because of the slow metabolism of thiopentone (10–15 per cent per hour) anæsthesia is prolonged. Anæsthesia depends not only on the concentration of the drug but also on the length of time of exposure of tissues to the drug. It rapidly crosses the blood-brain barrier and its concentration in the cerebrospinal fluid rapidly approaches that in the plasma. Equilibrium between plasma and brain is established one minute after intravenous injection. The initial high uptake of thiopentone by the brain accounts for the rapidity of the onset of anæsthesia.\* The effect of the drug on a particular patient depends on the plasma level at which equilibrium is established between the fat and the rest of the tissues. The other

\* Keele C. A., *Proc. R. Soc. Med.* 1952, 45, 215.

ultra short acting barbiturates—thialbarbitone hexobarbitone and thiamylal—act in a similar way

A decrease in the blood pH produced by carbon dioxide retention causes a fall in the plasma thiopentone level while a rise in the pH causes a rise in the thiopentone level\*. The greater the carbon dioxide retention the less the blood thiopentone level. Put another way a patient requires more of the drug if his carbon dioxide is allowed to accumulate and less if he is hyper-ventilated. Thus with a given dose of thiopentone the narcotic effect will last longer if he is hypoventilated than if his carbon dioxide is allowed to build up.

**RESPIRATORY SYSTEM**—The chief effect is depression of the respiratory centre depending on the dose and rate of injection. A deep breath or two or a yawn may precede the depression. It is because of the hypoxia produced by this depressant action that thiopentone may be dangerous from the respiratory view point. The depth of breathing is related to the external stimuli and a patient may breathe adequately on the operating table and then to subside into shallow respiration when left quiet and undisturbed in bed. Unless however respiration is greatly depressed the blood oxygen and carbon dioxide tensions undergo little change. If after respiratory arrest oxygen is supplied to the lungs the rapid destruction of the drug in the tissues will usually soon occur and spontaneous breathing will recommence. Under thiopentone the sensitivity of the respiratory centre to carbon dioxide is reduced or lost so this gas if in abnormal amount in the blood and tissues will act as a further depressant. On the other hand the carotid sinus and aortic body reflex response to oxygen lack resulting in respiratory stimulation is not so easily depressed as the respiratory centre. The Hering Breuer reflex to lung inflation is well marked during thiopentone anaesthesia i.e. apnoea lasting 5-30 seconds can be produced if the reservoir bag of an anaesthetic machine is compressed while the expiratory valve is closed at the end of an inspiratory effort. The deeper the anaesthesia the longer the apnoea. Preliminary hyperventilation prolongs it still further. This is a useful procedure during certain radiological examinations done on the operating table when a short period of apnoea is desirable. Reasons advanced to explain this phenomenon are (1) Thiopentone frees the lower reflex centre from cerebral inhibition (Gordh). (2) The drug blocks the central actions of respiratory afferents in the vagus contrary to what happens with ether or cyclopropane. (3) The drug depresses the respiratory centre so the stimulating effect of carbon dioxide, the build up of which during the apnoea no longer stimulates the respiratory centre.

Carbon dioxide should thus be used sparingly if at all to combat the hypopnoea due to overdosage of thiopentone.

The parasympathetic system is depressed less than the sympathetic by thiopentone and bronchial spasm is sometimes seen due

## INTRAVENOUS ANÆSTHESIA

### pentone Sodium—Pharmacology continued

to this cause as well as coughing and bucking. Coughing and mild laryngospasm may occur spontaneously after injection of thiopentone.

**CARDIOVASCULAR SYSTEM**—Rapid injection of too much thiopentone may have a most grave effect on the circulatory system. The force of the heart's contraction is weakened and the heart dilates but this is of no importance in normal hearts in fit patients. There is need for care when the drug is used in severe cases of cardiac decompensation as any existing coronary narrowing will result in a decrease of myocardial blood supply. The blood pressure is depressed—depending on rate and amount of drug injected—it is probably due to dilatation of the vascular bed perhaps due to depression of the vasomotor centre and the effect usually passes off with continuance of the anæsthesia. Depression is greater in hypertensive than in normotensive patients. Phenylephrine and methoxamine well diluted added to the thiopentone solution will prevent this depression and an even better remedy is 50–75 µg of noradrenaline ( $\frac{1}{2}$ – $\frac{1}{4}$  ml of 100 µg per 2 ml ampoules). In cases of hypertension the fall in blood pressure may be considerable a fact sometimes used to determine the suitability of such a case for sympathectomy. Thiopentone gives no protection from vagal inhibition of the heart which may follow the use of chloroform trilene or cyclopropane.

M. Johnstone points out that during thiopentone anæsthesia hypercapnia due to respiratory depression may cause ventricular ectopic beats to arise which disappear again when the carbon dioxide excess is removed. It has been suggested by Dundee\* that changes in plasma potassium content may result from hypercapnia and this is one cause of arrhythmia. Pre-existing ventricular extrasystoles are usually abolished by the drug. auricular ectopic beats e.g. auricular fibrillation are uninfluenced.

**LARYNX**—Spasm due to the parasympathetic effect is sometimes produced during thiopentone anæsthesia either by stimuli from the site of operation or from stimuli of the vagal nerve endings in the larynx by mucus blood airways etc. Spasm may in some cases be due to regurgitation of acid gastric contents even in small amounts (M. Johnstone). It has also been suggested that the region of the hypothalamus controlling the parasympathetic recovers before that part controlling the sympathetic system. This effect is lessened by the pre-operative administration of atropine or scopolamine parasympathetic depressants. Laryngeal irritability is less after hexobarbitone and thiamylal than after thiopentone.

**EYES**—Pupils first dilate then contract. Sensitivity to light remains until the patient is deep enough to permit incision of the skin and at this stage the eyeballs are usually centrally

placed. During surgical anaesthesia the corneal conjunctival eyelash and eyelid reflexes disappear. It reduces intra ocular tension.

**IRRICITANT UTIRUS**—Thiopentone has no effect on its tone and so is a poor agent used alone for external version.

**VALLOIAN TUBES**—No effect on tone of normal or abnormal tubes.

**MYONEURAL JUNCTION**—A mild curariform effect much less than that of ether.

**SPLEEN**—Probably not caused to dilate nor is hemodilution caused.

**ALIMENTARY CANAL**—Tone and motility only depressed after big doses.

**EXCRETION AND METABOLISM**—The liver probably breaks down the drug oxidizing its side chains. Between 10 and 15 per cent of the drug in the body is metabolized each hour. Muscular tissue may help in its detoxication as may the kidneys. It is doubtful if the drug causes any damage to normal livers but may well damage a handicapped or diseased liver and in patients so afflicted dosage must be small. The degree of liver dysfunction must be considerable before a patient shows diminished tolerance to thiopentone and tolerance is decreased only to intermittent doses given over a long period. The products of its breakdown are removed in the kidneys, but renal disease is not a contra indication to its use although a uremic patient will require smaller amounts than a normal patient. Urinary secretion is decreased during thiopentone anaesthesia.

**MISCELLANEOUS EFFECTS**—It passes into the cerebrospinal fluid and into the breast milk shortly after injection. It readily passes the placental barrier achieving its maximal concentration in foetal blood very soon after its injection into the mother. The intracranial and cerebrospinal fluid pressure falls, while the blood sugar and blood urea are not greatly affected. Disturbances in acid base equilibrium are likely to be due to hypoventilation—not to metabolic acidosis. A high blood urea prolongs thiopentone narcosis and the drug may not be the safest agent used alone for some patients undergoing cystoscopy. The barbiturates have an antagonistic action to cocaine and its derivatives (Tatum and others 1925). In some patients a localized muscular spasm is seen following injection. It usually takes the form of pronation of the forearm receiving the injection. Addition of nitrous oxide and oxygen usually controls it. It occurs more frequently with 5 per cent solution than 2½ per cent solution and can be lessened by opiates and pethidine.

Skin rashes have been very occasionally reported following its use. They may be scarlatiniform or urticarial.

It is a pure relaxer of the muscles of the anterior abdominal wall but usually entitles a gynaecological bimanual examination to be carried out satisfactorily and is sometimes used in small amounts to give just a little extra relaxation closure of a laparotomy wound.



**Thiopentone Sodium** *continued*

**Premedication** —At the time of operation the stomach bowels and bladder should be empty

Atropine or scopolamine should always be given to depress the vagal reflexes. An opiate is not necessary in short operations but is beneficial in longer ones for the following reasons —

- 1 It tranquilizes the patient and reduces his metabolic rate
- 2 It reduces the amount of thiopentone needed and so reduces the period of post operative depression
- 3 It helps to produce a smooth anæsthesia
- 4 It makes venepuncture easier

*Omnopon*  $\frac{1}{2}$  gr with *scopolamine*  $\frac{1}{4}$  gr is a favourite pre medication. Morphine  $\frac{1}{4}$ – $\frac{1}{2}$  gr with atropine  $\frac{1}{8}$  gr may be substituted. Some workers including the author prefer half these amounts. These injections should be given three-quarters of an hour before operation.

*Pentobarbitone* 3 gr by mouth two hours before operation and atropine  $\frac{1}{8}$  gr hypodermically three quarters of an hour before operation are also a good combination. The use of thiopentone does not contra indicate the use of pentobarbitone in reasonable dosage as a pre anæsthetic sedative.

**Course of Anæsthesia** —When an intravenous anæsthetic is given the following should be at hand in case of need (1) A laryngoscope (2) Endotracheal tubes (3) Oxygen (4) A mask and reservoir bag (5) A tilting table (6) Suction apparatus. Guedel's classification of stages of anæsthesia does not apply. The patient is either too light (he reacts to surgical stimuli) he is properly anæsthetized or he is too deep (his respirations are very shallow his blood pressure depressed). The first injection should be 4–8 ml of 2½ per cent solution and it can be made quite rapidly in fit subjects more slowly in others. The concentration of the drug reaching the brain in arterial blood immediately after injection is determined by the rate of injection. If the patient counts at a uniform rate an indication of his reaction to the drug is obtained. Just after the onset of unconsciousness there is often a deep breath followed by a period of respiratory depression. During this period no further injection should be given. With normal breathing re established further thiopentone is given according to the needs of the patient. If a little is injected at frequent intervals observing its effects no danger is likely to be encountered. It may be necessary to wait for surgical stimulation before the depth of anæsthesia can be ascertained for sure. A slight movement caused by the skin incision will often completely disappear once this severe stimulus has ended and is not necessarily an indication for deepening anæsthesia.

The criteria of depth are —

- 1 The activity of respiration in relation to surgical stimuli
- 2 Reflex movements of the patient in relation to such stimuli. When stimuli are severe depth may have to be increased e.g. when skin is incised or sutured.

Doses of thiopentone required vary from 0.1-2 g. Seldom should a larger dose be injected and if 2½ per cent solution is used rarely is 1 g. exceeded. A small dose in a fit patient results in a short period of narcosis but larger doses may be followed by prolonged sleep. Additional doses within thirty-six hours cause cumulation. A patient may respond to face slapping in the theatre only to return to the quiet and warmth of his bed and there meet death from an unobserved respiratory obstruction. Thiopentone anaesthesia except in the shortest and most minor operations should always be accompanied by nitrous oxide and oxygen. Six litres a minute of the former and 2 litres of the latter is a good mixture. It enables the depth of respiration to be accurately assessed, reduces the amount of thiopentone needed by about 50 per cent and guards against hypoxia.

A trace of trilete given before the pharyngeal airway is inserted will frequently depress coughing and gagging reflexes but should not in the author's opinion be used routinely.

Control of the airway is of primary importance in intravenous anaesthesia. Before the injection is started a dental prop or Baker's airway should be inserted between the patient's teeth so that a gag can be inserted atraumatically should spasm of the jaw occur in association with respiratory obstruction. A wisp of cotton wool held over the nares by a filament of adhesive strapping—a modification of Lundy's butterfly—gives a useful indication of the presence, rate and depth of breathing in those few cases not receiving nitrous oxide and oxygen. In many cases respiration is free if the head is fully rotated to one side. In others the lower jaw must be held forwards to draw the base of the tongue away from the posterior pharyngeal wall. Occasionally a pharyngeal airway or a nasopharyngeal tube is necessary but these should only be used if the airway becomes obstructed without them as they may stimulate pharyngeal reflexes which upset the smooth course of the anaesthesia.

Respiratory obstruction and relative overdosage resulting in apnoea causes hypoxia which added to the depressing effect of the barbiturate may soon inactivate the respiratory centre. The remedy for this respiratory depression or arrest is to get a free airway and to force oxygen into the lungs by pressure on the oxygen filled reservoir bag of a gas machine with expiratory valve shut, direct mouth-to-mouth insufflation via a face mask or by manual pressure on the thorax. Analeptics such as picrotoxin and nikethamide are of very secondary importance. When respiratory obstruction results from stimuli applied at too light a level of anaesthesia the surgeon should if necessary be asked to hold his hand until control of the airway is regained. The intravenous injection of a short acting relaxant e.g. succinylmethonium will also abolish spasm of the larynx due to this cause. Patients vary greatly in their requirements of the drug to abolish reflex response to stimuli. Males need more than females, the fat need more than the thin, the young need more than the old.

**Thiopentone Sodium continued**

**Recovery from Anæsthesia** —Rate of recovery is influenced by the amount of premedication and the amount of thiopentone injected. Post-operative restlessness is rare and vomiting is infrequent. During the immediate post operative period the airway and the tidal exchange must be carefully watched. Out patients should always be accompanied home after thiopentone anaesthesia.

**Agents used to supplement Thiopentone**

- 1 **GAS AND OXYGEN** —When given a 50 or 75 : 25 mixture full oxygenation is assured and less thiopentone is used. It is a safety measure and is used routinely in many clinics when thiopentone is the anæsthetic for all but short operations.
- 2 **MORPHINE** —Intravenous injection of  $\frac{1}{4}$ – $\frac{1}{2}$  gr is often helpful if a patient is taking much more thiopentone than average to obtain a level of smooth anæsthesia. After such a supplementary injection further thiopentone will often produce a profound effect.
- 3 **CYCLOPROPANE ETHER AND TRILENE** —It is often wise to add one of these agents if a sufficiently deep plane of anæsthesia is not readily obtained with average doses of thiopentone. To push thiopentone may result in respiratory depression and prolonged post-operative sedation. Minimal trilene with gas and oxygen makes a specially useful supplement with a powerful depressant action on vagal reflexes.
- 4 **PETHIDINE** —Serial injections of pethidine e.g. 20 mg may be used. This drug is a good analgesic but may cause as much respiratory depression as thiopentone if given too liberally.
- 5 **RELAXANTS** —Most useful to prevent such reflexes as laryngeal spasm associated with e.g. anal operations, skin incisions, perineal operations. Frequently used to increase the muscular relaxation which is not well marked when unsupplemented thiopentone is used.
- 6 **LOCAL ANALGESIA** —A little local analgesic solution injected into the skin before its incision and again before its re-suture will enable smaller doses of thiopentone to be used e.g. in the Trendelenburg operation for varicose veins in young men and in hæmorrhoidectomy etc. Topical analgesia of the pharynx and larynx as by an amethocaine lozenge and a spray will often prove useful before thiopentone anaesthesia in patients who are likely to need a pharyngeal airway.

**Complications of Thiopentone Anæsthesia —**1 **LOCAL —**

- a **PERIVENOUS INJECTION** —This may cause pain, redness and swelling. Rarely ulceration. It may lead to median nerve injury if injection is made into the medial side of the ante-cubital fossa. These symptoms are less severe when the 2½ per cent solution is used. Should solution be deposited out side the vein 10 ml of 1 per cent procaine can be injected into the area. This dilutes the thiopentone solution and by promoting vasodilatation aids absorption. The injection of 300 turbidity reducing units of hyaluronidase dissolved in 10–20 ml of saline also aids absorption and eases pain.

- ✓ **b LARYNGEAL SPASM**—This may result from (1) Direct local stimulation by an airway saliva blood etc (2) Stimulation of some remote area (Brewer Luckhardt reflexes) (3) Part of a general anoxic spasm Thiopentone predisposes to laryngeal spasm Depth of anaesthesia should be slowly increased and oxygen administered under pressure Endotracheal intubation may be required as a last resort but it may be very difficult and cause trauma If the spasm persists it may be wise to change over to another anaesthetic agent such as gas oxygen and trilete Intravenous injection of suramethonium together with oxygen under positive pressure may be required to relax the spasm Occasionally as in certain cases of asthma a dose of thiopentone may cause intensive bronchial spasm muscular rigidity and reflex apnoea—an alarming condition which usually yields to the intravenous injection of a rapidly acting relaxant together with oxygen given under positive pressure
- ✓ **c COUGHING**—Depth should be gradually increased Nitrous oxide-oxygen trilete or other agents may be required in resistant cases Hiccup occasionally seen
- ✓ **d SNEEZING**—May occur in eye operations in spite of adequate topical analgesia of the conjunctival sac and nasolacrimal duct Interruption of surgical stimuli with slow increase of depth of anaesthesia will often control this distressing symptom
- ✓ **e SALIVATION**—May be severe and may require the use of a sucker if artificial airways are used in the absence of proper atropine premeditation
- ✓ **f POST-OPERATIVE VERTIGO AND DISORIENTATION**—Because of the possibility of this condition out patients should be accompanied home
- ✓ **g TRUE CUTANEOUS ALLERGY** can occur either in the form of a scarlatiniform rash or as true angioneurotic oedema Frankis Evans and his colleagues suggest that the sulphone radical may be the cause They also report photosensitivity to thiopentone in patients recently exposed to sunlight
- ✓ **h INFECTIVE HEPATITIS**—Transmission of the virus of infective hepatitis with its twelve weeks incubation period if apparatus is not properly sterilized by autoclaving or the hot air oven

### Advantages and Disadvantages of Thiopentone Anaesthesia —

The advantages are —

- ✓ 1 Ease and rapidity of induction
- ✓ 2 Absence of stage of delirium
- ✓ 3 Rapid recovery and relative freedom from vomiting and post operative discomfort etc
- ✓ 4 Absence of respiratory irritation
- ✓ 5 Ability to increase depth rapidly

The disadvantages are —

- ✓ 1 Respiratory depression
- ✓ 2 Laryngeal irritability

**Thiopentone Sodium—Advantages and Disadvantages continued**

- ✓ 3 Poor abdominal relaxation with safe dosage
- ✓ 4 Circulatory depression in poor risk patients

**Indications**—These are legion almost every operation in surgery having been performed under intravenous barbiturate anæsthesia. It is specially useful—

- 1 For induction of general anæsthesia
- 2 For short operations
- 3 Under Service conditions where portability and relative ease of administration are advantages
- ✓ 4 For supplementing regional analgesia
- ✓ 5 In the presence of a cautery
- 6 For controlling convulsions during general or local anæsthesia eclampsia etc
- 7 For narco analysis in psychiatry and for electroconvulsive therapy
- 8 As a senior partner in the well established firm of thiopentone gas oxygen pethidine and relaxant

Thiopentone is a suitable anæsthetic for the following—

- 1 Certain eye operations Intra ocular tension is reduced in both normal and glaucomatous eyes
- 2 Minor gynæcological operations and examinations under anæsthesia
- 3 Cystoscopies in patients who are not seriously uræmic
- 4 Hæmorrhoidectomy anal fissures etc (usually supplemented by a relaxant)
- 5 Orthopædic manipulations
- 6 Encephalography and lumbar puncture under general anæsthesia
- 7 Producing abdominal relaxation quickly for a relatively short time during an operation under inhalation anæsthesia e.g. for suturing the peritoneum when the surgeon wants a little extra relaxation
- 8 For major surgery when the balanced combination of thiopentone gas-oxygen pethidine and a relaxant are used

**Contra indications**—Opinions differ as to contra indications some workers denying that any absolute contra indications exist

In the following procedures and types of case special care is needed and oxygen or nitrous oxide-oxygen half of each must be given in addition—

- ✓ 1 Children under 4 because while their respiratory centres are easily depressed their upper respiratory passages are relatively small these factors predisposing to hypoxia Moreover children do not like needles while their active reflexes require large amounts of thiopentone for their subjugation so that they sleep for long periods after operation
- ✓ 2 Shocked debilitated severely anæmic and uræmic cases small doses are required The administration of pure oxygen is useful in handicapped patients before anæsthesia is induced It also aids the removal of nitrogen from the lungs and allows nitrous oxide to exert its analgesic effects more quickly so reducing the need for thiopentone

- There may be a dose of the drug small enough for induction of anaesthesia with safety in even the most decrepit patient but in the gravely ill cyclopropane has some advantages
- ✓ 3 Patients with gross dyspnoea due to cardiac or respiratory disease It should be used with extreme caution—if at all—in cases of constrictive pericarditis (Parry Brown) Possibly
  - 4 Patients with respiratory obstruction
  - 5 Operations in which the return of reflexes immediately after operation is desirable It is difficult to have a patient adequately anaesthetized one minute and coughing the next e.g. in tonsillectomy
  - ✓ 6 In bronchoscopy and oesophagoscopy—unless adequate topical analgesia and/or a relaxant is used also Dangerous laryngeal spasm may be initiated
  - ✓ 7 Cases of acute intestinal obstruction Aspiration of regurgitant vomit may cause dangerous laryngeal spasm unless proper care is taken to prevent it Also the liver in such cases is often functioning badly
  - ✓ 8 Patients with acute inflammation about the mouth jaw and neck Several deaths have occurred under thiopentone anaesthesia in such patients A likely cause of death is interference with the airway associated with spasticity of the jaw due to inflammatory oedema
  - 9 Asthma Attacks have occasionally been made worse by thiopentone in other cases no adverse effect has been seen The drug should be given slowly the anaesthetist feeling his way cautiously Better still soluble hexobarbitone should be used instead
  - 10 Feeble elderly patients on the brink of dementia Recovery may be slow and associated with disorientation
  - ✓ 11 For external version, thiopentone is a poor relaxant For delivery no more than an induction dose should be used (0.25 g)
  - 12 In malarial patients a little goes a long way
  - 13 Cases of dystrophia myotonica (J W Dundee) The patients react normally to curare but abnormally to thiopentone\*
  - 14 Addison's disease and myxoedema Must be used with great care
  - 15 Myasthenia gravis Great care necessary
  - 16 Patients with hepatic dysfunction Small doses only
  - ✓ 17 Porphyria† which may be congenital or appear as acute attacks of abdominal pain somewhat resembling lead poisoning with the passage of reddish urine Barbiturates may precipitate lower motor neurone paralysis and perhaps death and are absolutely contra indicated If suspected the urine should be tested for porphyrins
  - 18 Alcoholics taking disulphuram (antabuse) and patients suffering from poisoning with dinitro-ortho-cresol a weed killer barbiturates and D N C have a synergistic depressant effect on cellular respiration ‡

Lodge A B *Brit med J* 1958 1 1043

† Dundee J W and Riding V E *Anaesthesia* 1955 10 33

‡ Edson E F and Carey F M *Brit med J* 1955 1 104

**Thiopentone Sodium—Contra indications** *continued*

- 19 Patients with difficult veins There is no excuse for subjecting a patient to the painful experience of multiple needle punctures in an effort to provide a pleasant induction of anæsthesia

**Other Methods of Thiopentone Administration.**—Various types of equipment have been developed to enable thiopentone to be delivered to a vein some by remote control

Solution of the normal strength can be injected into the tubing of an intravenous drip by a fine intradermal needle It can be dissolved in saline in strengths of 0.1 to 2.5 per cent and run in as a drip Each 0.5 g of thiopentone added to a pint bottle of intravenous fluid increases the percentage roughly 0.1 per cent ✓ Thus 0.5 g to 1 pint is 0.1 per cent 1 g to 1 pint is 0.2 per cent etc A drip rate of 40 per minute roughly equals 140 ml per hour or 1 pint in 4 hours 5 per cent dextrose in water can be used A useful method is to interpose a three way tap between the syringe and needle and lead a saline drip to the third arm of the tap Thus the drip ensures a patent needle and if the tap is turned thiopentone solution is directed from the syringe into the vein

**Description of Average Thiopentone Administration**—The arm of the patient is abducted placed on an armboard and built up with pads underneath so that no strain is placed on the brachial plexus A piece of rubber tubing is fixed around the upper arm with a hæmostat and the presence of the radial pulse is verified a second rubber tube is likewise fixed with a hæmostat surrounding



Fig. 47.—The Franks Evans intravenous needle.  
(Medical & Industrial Equipment Ltd.)

the wrist and armboard A vein is selected preferably one in the long axis of the limb It is cleaned with antiseptic and a small weal is raised just lateral to the vein with the finest intradermal needle using 1 per cent procaine solution Through the weal a larger needle is inserted (e.g. No. 1) and it is advanced into the vein as great a distance as it will safely go The insertion of a Mitchell or other non clotting needle through the skin weal is a preferable and more elegant technique for the experienced worker Thiopentone solution 2½ per cent is injected after the anaesthetist is certain that his needle is truly intravenous this may need aspiration of a little blood into the syringe 0.05 g (2 ml) is injected and the patient is asked if his arm is comfortable Should pain occur in the fingers or hand it indicates that the injection is probably intra arterial in which case harm is unlikely if no more is given In the absence of hand pain a further 5–10 ml is injected and when the patient loses consciousness if a non clotting needle has not been used the tourniquet tubing is used

to strap the syringe on to the arm it encircles syringe arm and armboard and is secured with an artery forceps not too tightly for fear of injuring the median nerve. Nitrous-oxide-oxygen are now given (50:50 or 75:25) so that a total flow of 7-8 litres a minute is available. Just before the surgical stimulus is given more thiopentone is injected the amount depending on the type of patient his reflex irritability and the type of operation. Minimal trilene can usefully be added. (1) If the patient coughs gags or bucks. (2) If the insertion of an airway being necessary is likely to disturb the smooth anaesthesia. (3) If reflex activity (lightness) reappears after 0.75 g of thiopentone has been injected—to prevent use of too much barbiturate. Pethidine can be used for the same purpose. (4) If an endotracheal tube is used—to prevent coughing. (5) If it is important for the patient to recover consciousness early. Trilene will slightly raise the incidence of vomiting. The addition of repeated small amounts will also quieten upper respiratory tract reflexes.

Automatic electroencephalographic control of thiopentone anaesthesia has been described\*.

**Drugs Chemically Incompatible with Thiopentone**—Pethidine, alphaprodine, phenothiazine derivatives, laudexium, methyl sulphate, suxamethonium salts, nalorphine, procaine hydrochloride, papaverine.

The following drugs form with thiopentone a slight precipitate which redissolves in an excess of the thiobarbiturate: papaveretum, morphine, levorphan, pentolinum tartrate, trimetaphan, methylamphetamine, methoxamine, levallorphan, lignocaine, pitressin.

### OTHER INTRAVENOUS ANAESTHETICS

**Buthalitone Sodium** (Transthal, Baytenal, Vibrevall, Thialbutone)—This is the sodium salt of 5-allyl-5<sup>1</sup>-isobutyl barbituric acid and was synthesized in 1936 by Müller et al. in the U.S. and investigated by Weese and Koss in 1954 in Germany. It is used in 5 or 10 per cent solution and its effects are said to be less prolonged than those of thiopentone while the post-anaesthetic confusion and drowsiness is also said to be less. Has been used to precede nitrous oxide and oxygen in the dental chair (400-600 mg)† and in the casualty department‡. Useful together with a relaxant for manipulations (1,000-1,000 mg). Weight for weight about half as potent as thiopentone.

**Soluble Hexobarbitone (B.P.)** (Evipan, Evipan soluble, Cyclonal, sodium Hexanostab, Hexanol, Oevipan, Oulapan)—This is sodium N-methyl-c-c-cyclohexenyl methyl barbiturate. First used in 1932 by Weese and Scharpf after being synthesized by Kropp and Taub.

It is used as a 10 per cent solution (though 5 per cent is probably preferable) its action being similar to though less potent, less depressing to the respiration and circulation and less irritating to the tissues than thiopentone sodium. Twitching, sneezing,

\* Jersey D. K., Faulconer A. jun. and Bickford, R. G. *Anesthesiology* 1954 15 356

† yk. B.D. *Anaesthesia* 1957 12 259

‡ Young D. S. *Proc R Soc Med* 1956 49 735

§ Oles P. *Lancet* 1955 1 797



**Soluble Hexobarbitone** *continued*

etc are more frequent during induction than with thiopentone but respiratory depression and bronchial and laryngeal spasm are less common. Average dose for induction of anæsthesia 400 mg

**Thialbarbitone** (Kemithal) — Sodium 5-cyclohexamyl 5 allyl 2 thio barbiturate. Prepared by Carrington in 1938 in Britain but not investigated pharmacologically until 1946\* (Carrington and Raventós). Supplied in ampoules of 1 and 2 g of dried powder. Like thiopentone it contains a sulphur atom. Used as a 10 per cent solution which remains stable for less than six hours. In anæsthetic effect kemithal 1 g equals thiopentone 0.5 gr. Kemithal is less likely to produce laryngeal spasm and respiratory depression than an equivalent anæsthetic dose of thiopentone. During recovery especially in children generalized muscular rigidity unassociated with laryngeal spasm may result from any strong stimulus.

**Pernocton** — A 10 per cent solution of sodium butyl betabromallyl barbiturate. First used by Bumm in 1927. The first widely used intravenous barbiturate in anæsthesia.

Excretion too slow for safe routine use but it can be used for induction 1 c.c. being given each minute until the patient is asleep. As it is stable in solution it is supplied ready dissolved.

**Pentobarbitone** (Nembutal Pentobarbital) — First used intravenously by Lundy in 1931. It is less likely to cause laryngeal spasm than is thiopentone and has been used before endotracheal intubation on this account. Useful to produce sleep during operations under regional analgesia. Usual strength 0.75 gr per ml (7½ per cent). Patient wakes up less confused than after thiopentone. Sodium salt used for intravenous injection.

**Quinalbarbitone** (Sodium seconal) — Has a longer though less intense action than thiopentone. Has no analgesic effect. May be useful for producing sleep during a good regional block. Laryngeal spasm not very frequent.

**Thiamylal** (Surital sodium Thioquinalbarbitone Thioseconal sodium) — First described in 1935 by Volwiler and Tabern. Sodium 5 allyl 5 (1 methylbutyl) 2 thiobarbituric acid. It is the sulphur salt of quinalbarbitone (seconal) and is used in 2½ per cent solution. Very similar in its action to thiopentone slightly greater in potency and with less tendency to cause cumulation. Many barbiturates give motor and sensory block when injected intrathecally.

**Intravenous Ether** — Used in animals by Pirogoff in Russia in 1847 a year after Morton's use of ether by inhalation. Used in surgery by Burckhardt in 1909.

A 2½ to 5 per cent solution in normal saline is used. 12.5 to 25 ml of ether being well shaken up in a bottle containing 500 ml of saline warmed to about 90° F. not more as the boiling point of

ether is 93.6° F. After complete solution a continuous intravenous drip is set up slowly at first and later the fluid running as a steady stream. Once anaesthesia is induced which may require 400 ml of solution or more maintenance rate is 40-60 drops per minute. Signs of anaesthesia are similar to those when ether is given by inhalation although respiration is quieter. Premedication is as for ether given by inhalation.

Vomiting and respiratory irritation are less than with inhalation ether anaesthesia. A solution stronger than 5 per cent may cause hæmoglobinuria the method is contra indicated in renal disease. The airway needs the same attention as with any general anaesthetic. May cause thrombo phlebitis.

Recovery is fairly rapid.

**Hydroxydione Sodium Hemisuccinate (Viadril Presuren) \***—This is 21 hydroxy pregnane 3, 20 dione sodium succinate and is a steroid a group of agents never used before to produce anaesthesia. First used by Murphy of the University of California in 1955 at the suggestion of Laubach and his colleagues. It is a crystalline solid sold in 0.5 g ampoules. Because of its tendency to irritate veins it should be given in 0.5 per cent solution in dextrose as a continuous drip running into a large limb vein. At a drip rate of 150 per minute the patient falls asleep in 10 to 20 minutes. Its solution is incompatible with solutions of pethidine, d-tubocurarine and suxamethonium. It has no endocrine activity. It finds its chief use together with premedication, pethidine and gas oxygen and should not be used as the sole anaesthetic agent. When 500-700 mg have been run in to the patient laryngo copy can usually be performed without causing pharyngeal and laryngeal reflex activity. Post anaesthetic drowsiness may last for an hour or two but vomiting is rare and the patient is said to feel better than after a comparable dose of a thiobarbiturate. Abdominal relaxation poor, hypotension sometimes caused, effect on myocardium not usually serious although tachycardia has been reported. Respiratory depression less than after thiopentone. Its disadvantages are its tendency to cause thrombophlebitis and the slow onset of anaesthesia. One advantage is its benign effect on the laryngeal reflexes. Such complications as fall in blood pressure and respiratory depression may not occur until some time after the administration of the drug has stopped †. Presuren may be kinder to vein walls than Viadril if given intravenously in one half to two minutes in 2.5 per cent solution in warm saline. Dosage 5-8 mg per pound. A large vein should be used and additional drugs should not be injected into this vein. It is recommended for the induction of anaesthesia in some cases of intestinal obstruction and in thyroidectomies ‡.

**Intravenous Paraldehyde**—This method was used first in 1913 by Noel and Souttar. Paraldehyde has very little respiratory depressing effect and in normal doses does not damage the liver. If a

\* Maitto, G. A. and others *Anesthesiology* 1958 19 450.

† Hunter, A. R. *Anaesthesia* 1957 12 19. Dent, S. J. and Wilson, W. I. *Anesthesiology* 1956 17 673.

‡ Landau, E. *Anaesthesia* 1958 13 247.

**Soluble Hexobarbitone continued**

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‡ Landau E. *Anaesthesia* 1958 13 247

**Intravenous Paraldehyde continued**

concentrated solution is injected quickly cough and laryngeal spasm may be produced and doses of 2 ml have been given intravenously to stimulate coughing after operation. It has been given as a 6-8 per cent mixture with isotonic glucose at the rate of 15-20 ml each minute. It has also been mixed with saline and ether. Paraldehyde must be fresh. To sterilize it it can be autoclaved at 130 C for 30 minutes without decomposition. As a 2½ per cent solution in normal saline intravenously it acts as a good anticonvulsant dosage 0.2 ml per kg of body weight. It depresses the cerebral cortex.

**Intravenous Morphine.**—The intravenous injection of morphine produces a rapid effect. It is useful —

- 1 In acute pain especially if associated with shock. Shock makes absorption from subcutaneous tissues slow and uncertain. On its reversal sudden absorption takes place.
- 2 Before regional analgesia if patient is ill at ease.
- 3 To supplement regional analgesia for painful examinations e.g. bronchoscopy.
- 4 To premedicate patients before emergency operations.

Morphine should be given slowly in dilute solution in similar dosage to that used for hypodermic injection. Like thiopentone morphine may increase laryngeal irritability.

The respiratory depressant effects of morphine (and pethidine) can be readily controlled by amiphenazole. Nausea and vomiting by cyclizine and constipation by senokot.\*

**Intravenous Alcohol** — This was reported on by Marin of Mexico in 1929.

The strength of 30 per cent alcohol in isotonic glucose has been used the rate of injection being 20 c.c. per minute. Anaesthesia may take 15-20 minutes to come on.

Thrombosis of the vein is the chief drawback.

**Intravenous Avertin.**—First used in 1929 by Kirschner. This can be given as a 1 per cent solution in saline as a continuous drip. It is not so likely to cause laryngeal spasm as thiopentone. It causes sleep without restlessness and dilates the bronchi. It reduces the blood pressure. Avertin 5 ml can be added to 500 ml of 5 per cent dextrose and well shaken up. The drip should run rapidly and sleep comes on after 75 to 250 ml have run in. Can be used to maintain sleep during regional analgesia and is useful together with topical analgesia for bronchoscopy and oesophagoscopy. It is contra-indicated in liver disease. Causes thrombophlebitis in about 6 per cent of cases.

**Intravenous Procaine** — First used in 1909 by Bier who employed a tourniquet to localize solution in a limb. Later used to relieve pain in endarteritis obliterans (Leriche and Fontaine 1935) to reduce tinnitus aurium (Bárány 1935) to relieve the itchiness of jaundice (Lundy 194) and to make dressing of burns painless.

(Gordon and Tovell 1943) McLachlin used it for post-operative pain (1945) Its steadying effect on the heart was investigated by Rovenstine and Burstein in 1940

**CHEMISTRY** —Procaine belongs to the alkaline ester group of drugs which includes atropine tubocurarine and the antihistamines Solutions can be sterilized by boiling but this reduces the pH so that solution becomes more acid more irritating and less efficient as an analgesic As solutions tend to oxidize on keeping with alteration of pH solutions should be freshly made

**PHARMACOLOGY** —Injected intravenously it paralyzes first the parasympathetic later the sympathetic synapses and curarizes voluntary muscle motor end plates perhaps by inhibiting the release of acetylcholine these effects being antagonized by neostigmine and diisopropylfluorophosphate On the other hand procaine protects against most symptoms of neostigmine and D F P toxicity Procaine and neostigmine are antagonistic procaine and curare are additive The tachycardia occasionally caused by procaine will yield to intravenous neostigmine 0.5 to 1 mg

On the central nervous system it causes muscular twitching anxiety disorientation and finally convulsions The last are treated by stopping administration and injecting small doses of intravenous barbiturate together with administration of oxygen and if necessary drugs to raise the blood pressure

Reduces the irritability of the automatic conductive tissue of the heart and has a quinidine like effect on the auricle It causes low blood pressure It has an antihistamine action and antagonizes acetylcholine When injected intravenously it becomes concentrated in oedematous traumatized and inflamed tissue owing to the increased capillary permeability in these situations It is hydrolysed partly in the liver and partly in the blood stream by an enzyme into para aminobenzoic acid and diethylamino-ethanol being one of the most rapidly metabolized drugs known It is excreted by the kidneys—2 per cent as procaine 28 per cent as diethylamino ethanol and 70 per cent as para aminobenzoic acid Some of the last combines with glycine to form aminohippuric acid and some is conjugated with glucuronic acid The comparative safety of the procaine drip lies in this rapid breakdown The esterase which causes its hydrolysis is like acetylcholine inhibited by neostigmine It may act as a sensitizing agent causing contact dermatitis either in the form of (1) Allergic eczema or (2) Pompholyx The former disappears on withdrawal of the drug but the latter is not so easily cured After injection it causes on very rare occasions urticaria or angioneurotic oedema

True sensitivity is rare and is proved only if the intradermal injection of 1 ml of 1 per cent solution causes within ten minutes dyspnoea agitation and disorientation

Liver deficiency does not contra indicate its use but it should not be employed in patients with myasthenia gravis or in subjects receiving full doses of digitalis or one of its congeners

Intravenous Procaine—Pharmacology *continued*

Vitamin C deficiency together with fluid and electrolyte imbalance may increase toxicity. This can be overcome by giving glucose saline containing 1 g of ascorbic acid to each litre.

The optimal effective dose is 4 mg per kilo (25 mg per stone) as a 0.1 per cent solution given in twenty minutes—this is one procaine unit (Graubard). For emergency use 10 ml of 1 per cent solution can be given intravenously.

**CLINICAL USES**—It is not to day a drug which is used very frequently for intravenous injection.

- a To supplement general anaesthesia by thiopentone and gas-oxygen. After induction 1 per cent procaine in saline is given as a drip starting at 4 ml per minute later slowing down to half this amount. Signs of overdosage are twitchings which subside if the drip is stopped. About 2–3 g per hour are required by adults. The vasodilatation caused hampers the surgeon.
- b To inhibit cardiac arrhythmia during cardiac and thoracic surgery during cyclopropane anaesthesia etc. It is used less frequently than it was some years ago—it is after all a cardiac depressant.
- c In allergic states such as status asthmaticus serum sickness bronchospastic states during anaesthesia.
- d In doses of 5–10 ml of 1 per cent solution it increases the speed of intravenous drips (Organe and Scurr).
- e To produce analgesia during the dressing of burns and to control post operative pain without causing respiratory depression. Beecher\* has advised against its use for this purpose because of its inefficiency and undesirable side-effects.
- f To relieve certain painful trophic and vasomotor conditions frost bite phantom limb intermittent claudication etc.
- g To improve cases of amblyopia following retinal vascular occlusion together with stellate ganglion block. To relieve pain and reduce intra ocular pressure in acute glaucoma.
- h In treatment of intractable pruritus.
- i To relieve pain in chest injury (Rook†) where its use allows the application of strapping without the need for intercostal block. Cardiorespiratory embarrassment is relieved and wet lung and atelectasis are less likely to occur.
- j To relieve pain and spasm in acute anterior poliomyelitis.
- k To relieve orchitis and epididymitis.
- l To improve lower nephron nephrosis with its associated oliguria.
- m To relieve prolapsed piles.
- n To ease labour pains.

It can be combined with penicillin 250 units of the latter added to 1 ml of 1 per cent procaine solution.

It reduces the analgesic and relaxant effects of thiopentone-curare mixtures.

It should not be given along with neostigmine.

Beecher H and others *J Amer med Ass* 1951 147 1761  
 † Rook J R. *Anaesthesia* 1951 6 4

This whole subject is well discussed in Graubard and Peterson's book.\*

**Intravenous Lignocaine** —In 0.5 per cent solution this has been used to ease the pain of carcinomatosis and to relieve labour pains. It seems to be less toxic than procaine for a given degree of analgesia †. It appears that lignocaine is not broken down by pseudocholinesterase. The liver is however capable of breaking down the drug and diethylaminoacetic acid is one of the chief metabolites ‡. It potentiates the hypotensive effects of hexamethonium §. It has been used to produce analgesia during the thiopentone-gas-oxygen relaxant sequence when 40 mg are injected intravenously every five minutes. A dose of 500 mg should not be exceeded during the first hour. During the second hour the amount injected is halved. Twitching of the eyebrows the first sign of toxicity calls for cessation of injection together with a small intravenous dose of a barbiturate. Thus used it is said to give a useful degree of analgesia both during the operation and also in the post-operative period. It causes no appreciable circulatory or respiratory depression, a low incidence of post-operative vomiting and an early return to consciousness ||. *Has been used to control status epilepticus—200–400 mg in 1 per cent solution\*\**

**Procaine Amide Hydrochloride (Pronestyl, Procaryl)** —This is procaine with an amide grouping  $\text{CO NH}$  instead of an ester grouping  $\text{CO O}$ . It acts for longer periods than does ordinary procaine because it is not hydrolysed by pseudocholinesterase. It is active when given by mouth. It causes no central nervous stimulation and less hypotension than procaine but will produce nausea and vomiting and in rare cases pyrexia, agranulocytosis, allergic phenomena and even ventricular fibrillation. It is a very weak analgesic. Peak plasma level occurs one hour after oral, 15 minutes after intramuscular administration.

Used in cardiology as a substitute for quinidine to treat paroxysmal ventricular tachycardia and ventricular extrasystoles. It is not so useful for auricular arrhythmias. The refractory period as measured by the Q-T interval is prolonged and conduction is slowed. It is not without danger and latterly has been injected intramuscularly rather than intravenously. Excreted mostly unchanged by the kidneys, a small amount excreted as para-aminobenzoic acid.

In anaesthesia it is sometimes used to prevent ventricular arrhythmias arising during thoracic surgery and to reduce the incidence of arrhythmia during intubation. It potentiates hexamethonium and reduces tachycardia associated with ganglionic blocking agents. Its action in counteracting the arrhythmias associated with cyclopropane anaesthesia is inconsistent while it will not protect against the severe cardiac disturbances due to the

Graubard D. J. and Peterson M. C. *Clinical Uses of Intravenous Procaine* 1950 Oxford: Basil Blackwell.

† Gilbert, C. R. A., Hinson, R. A. and others, *Curr Res Anaesth* 1951 30 301

‡ Geddes I. C. *Anaesth* 1958 13 700

§ de Clive Low S. G., North, J. and Gray P. W. S. *Anaesthesia* 1954 9 96

|| — — — and Desmond, J. *Ibid* 1958 13 138

Tavener D. and Bain W. A. *Lancet* 1958 2 1145



**Procaine Amide Hydrochloride** *continued*

combination of cyclopropane and adrenaline. Should be used with care in asthmatics and is contra indicated during therapy with sulpha drugs.

By mouth (the preferred route in cardiology) it is given in capsules each containing 250 mg. The intravenous dose is 3 ml of 10 per cent solution given over a period of one to five minutes and cautiously repeated if required.

**Intramedullary Medication** \*—The bone marrow of the sternum or in infants of the tibia is a convenient place to infuse various fluids either for purposes of resuscitation or anæsthesia. In the absence of veins intramedullary infusion of fluid may save lives in acute shock.

The method was popularized by Tocantins in 1941. Once the needle is in place it will not easily slip out. (Fig 48)



Fig 48—Sabla's sternal puncture needle with adjustable stop (Medical and Industrial Equipment Ltd.)

**TECHNIQUE**—After scrubbing up the sternum is cleaned and towelled off. A weal is raised with 0.5 per cent novocain either over the manubrium or the upper part of the body of the sternum in the midline. Novocain is carried down to the pericostum. The skin is nicked with a tenotome and the special wide bore needle with stylet is passed down to the bone. The outer plate is pierced with a boring action, this movement being carefully controlled to prevent perforation of the posterior plate. The stylet is withdrawn, a syringe attached to the needle and marrow substance aspirated. This causes some discomfort. The presence of blood like marrow in the syringe indicates that the needle point is correctly placed. A few ml of sodium citrate solution is injected to clear the needle and a continuous drip apparatus is connected up. Occasionally gravity or a positive pressure is necessary to produce a steady flow. Blood, saline, dextrose, thiopentone in 0.5–2 per cent solution, avertin 1 per cent solution can all be given via this route. Sulphanilamide powder is sprinkled round the needle in the bone and a sterile dressing applied.

**Pethidine**—To recapitulate its pharmacological effects it has the following actions: (1) Morphine like effect on pain; (2) Papaverine like effect on smooth muscle resulting in relaxation of bronchi, intestine and uterus; (3) Atropine like effect on heart and salivary glands; (4) Antihistamine effect. It antagonizes the action of acetylcholine or smooth muscle.

Useful together with local analgesia for endoscopic examinations †

Dealt with here for convenience. Also see noted by Trow, A. B., Turkel, H. and Thompson, M. S. *Anæsthesiology* 957 13 507.  
† Markby, C. E. P. *Brit med J.*, 1958 1 1397

Intravenous pethidine during anaesthesia was first used in 1947 by Neff and his colleagues in the U.S.A. and by Mushin and Rendell Baker in 1949.\*

**TECHNIQUE**—Premedication may be either morphine and atropine omnopon and scopolamine or pethidine 100 mg and scopolamine  $\frac{1}{16}$  g. After a sleep dose of thiopentone (0.25–0.75 g) gas and oxygen are given and pethidine 10–25 mg injected intravenously. After a short period further pethidine (25 mg) is given and repeated in the same dosage roughly every half hour. It must be given before the patient reacts to stimuli not afterwards as it takes some time to act. A relaxant is given as required. If veins are bad 100 mg can be given intramuscularly as soon as the patient is asleep. Can be given to children in doses reduced in relation to their weight. Has been given as a continuous intravenous drip—100 mg to 500 ml—at a rate of approximately 1 mg per minute.

Pethidine has recently been combined with levallorphan (100 mg of pethidine with 1.25 mg of levallorphan) under the name of Pethulorphan. The levallorphan is stated to have an optimal effect on respiratory depression and a minimal effect on analgesia.

**ADVANTAGES**—It allows patients to be comfortably awake very soon after the end of the operation.

It relaxes the bronchi, allows easier pulmonary inflation and depresses upper respiratory tract reflexes sometimes seen after intubation and after thiopentone.

It reduces the rapid ventilation rate sometimes seen after even small amounts of tralene.

It postpones and reduces the need for post-operative sedation.

**DISADVANTAGES**—It may cause respiratory depression and slow breathing. But this can be counteracted by the injection of nalorphine or levallorphan.

It may cause hypotension and circulatory depression due to myocardial depression and also to vasodilatation and so anaesthesia should be started with a test dose of 10–25 mg. This effect is sometimes delayed for several hours.

It contracts the sphincter of Oddi and so may interfere with successful exploration of the common bile duct.

In cases of severe liver dysfunction smaller amounts of pethidine than normal are required to produce the desired therapeutic effect before, during and after operation †.

It may cause urticarial weals to overlie the vein into which it is injected. This is probably of no consequence.

**Intramuscular Analgesia**—Lignocaine has been given intramuscularly to potentiate analgesia during the thiopentone-gas-oxygen sequence. The dose recommended is 75 mg/stone in 2 per cent solution e.g. 250 mg before induction after induction and then hourly‡. It is said to increase tube tolerance.

\*Mushin, W. W. and Rendell Baker, L. *Brit. med. J.* 1949, 2, 472.

†Dundee, J. W. and Tinker, L. F. *Ibid.* 1952, 2, 703.

‡Dawkins, C. J. M. and Steel, G. C. *Anaesthesia* 1957, 12, 425.

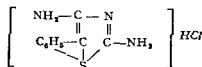
## CHAPTER XIV

## STIMULANTS, ANTAGONISTS, AND ANALEPTICS

(An = complete absence of, Lepsis = attack)

The chief action of these drugs is to stimulate respiration and to reverse narcotic activity of sedative drugs. When given in large doses they are mostly convulsants. Their stimulating action on respiration is greatest in non anaesthetized or lightly anaesthetized patients. In deep respiratory depression their effect is minimal and may be harmful. In conditions of respiratory arrest their use comes far behind that of oxygen.

**Amiphenazole**—This is 2,4-diamino-5-phenylthiazole hydrochloride and goes under the trade name of daptazole or DAPT, and was investigated by Shaw and Bentley in 1949\*. It is soluble in water but decomposes if allowed to stand for more than twenty-four hours (or up to seven days in a refrigerator). The dry powder is stable and it is usual to dissolve 30 mg in 15 ml of saline (or morphine solution). When injected into a patient whose breathing has been depressed by morphine, pethidine, alphaprodine, methadone, etc., considerable increase in the depth of breathing rapidly takes place. It has no effect on normal undepressed respiration.



In addition to counteracting respiratory depression in the morphinized patient, amiphenazole also: (1) Reduce vomiting, (2) Lessen constipation, (3) Restores the cough reflex. In clinical doses the drug apparently does not reduce the depth or duration of sensory depression. Given with bemegride it acts as a respiratory stimulant in cases of overdosage with barbiturates.

The use of the drug with morphine to control the depressant effects while allowing the analgesic effects to remain has been reported† and has given good result in the treatment of patients with chronic severe pain and in the alleviation of post-operative pain. An initial dose of  $\frac{1}{4}$  to  $\frac{1}{2}$  grain of morphine is given accompanied by amiphenazole 75 mg intramuscularly given from one syringe. This is repeated when the pain returns, the aim being to keep the patient completely free from discomfort. Should the rate of respiration be less than eight per minute or should any

Shaw F H and Bentley G M J *Aust J* 1949 2 868

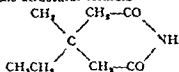
† McKeogh, V. and Shaw F H *Brit med J* 1956 1 143

Shaw F H, McCance L. and Bruce D W *Ibid* 1958 1 675

d Christ G Gray R

cyanosis appear 20 mg of amiphenazole is given intravenously and repeated at ten minute intervals until the respiratory depression has been overcome. The average patient shows no signs of toxicity from 200-300 mg of the drug. A rough guide as to dosage is to give equal amounts of amiphenazole and morphine.

**Bemegride** (Megimide N P 13) — This is  $\beta$ -ethyl  $\beta$ -methyl glutarimide and has the structural formula —



It was first used in the treatment of barbiturate overdose in the medical wards\* and after intravenous thiobarbiturate anaesthesia† in 1955.

It appears that the drug acts as a central cerebral stimulant and not as a specific barbiturate antagonist and so is unlike nalorphine in its reversal of morphine overdose but it alters the E.C.G. pattern of barbiturate intoxication. It does not increase the rate of excretion of barbiturate nor does it decrease the duration of coma but it produces a relatively safe state where respiration and reflexes are no longer depressed and when blood pressure, pulse rate and muscle tone are nearly normal. Untoward effects include vomiting and convulsive movements and a toxic psychosis resembling mescaline or lysergic acid intoxication. For the reversal of thiobarbiturate anaesthesia the intravenous injection of 10 ml of 0.5 per cent solution is recommended but larger doses may be required. Patients so treated may fall asleep again but can be thereafter readily aroused‡. Barbiturate intoxication is treated by the intravenous injection of amiphenazole and bemegride. The drugs are given into the tubing of an intravenous drip the amiphenazole as a 1.5 per cent solution the bemegride as a 0.5 per cent solution separate syringes being used. One ml of amiphenazole is followed by 10 ml of bemegride and these injections are repeated at five minute intervals until respiration and reflex activity return. As much as 20 ml of amiphenazole and 200 ml of bemegride may be required—10 to 20 injections.

It has been stated to reverse the hypnotic effects of the following drugs—doriden, dolitron, carbromal, chlorbutol, methyl pentynol, chloral hydrate, paraldehyde, hydroxydione§.

**Vanillic Acid Diethylamide** (Vandid) — This is a central respiratory stimulant. 10 ml of 5 per cent solution given intravenously to an adult has given good results in poisoning by barbiturates.

\* Shulman A. Shaw F. H. Cass N. M. and Whyte H. M. *Brit med J* 1955 1 1238 and Gershon S. and Shaw F. H. *Ibid* 1957 2 1509.

† Harris T. A. B. *Lancet* 1955 1 181.

‡ Wyne B. D. and Frayworth E. *Lancet* 1957 2 1025 and Waite T. E. and Dinmore P. *Anaesthesia* 1958 13 324.

§ Shulman A. and Laycock G. M. *Brit med J* 1958 1 871.

**Vanillic Acid Diethylamide** *continued*

morphine and coal gas For neonates the dose is 0.2 ml of 5 per cent solution intramuscularly

**Coramine** (Nikethamide B P Anacardone Corvotone and Nika mide) —Nicotinic acid diethylamide a camphor derivative put up in 25 per cent solution Allied to nicotinic acid

First synthesized in 1923 It stimulates the respiratory centre and the carotid and aortic bodies It slightly improves the circulation and produces peripheral vascular dilatation but has no direct stimulant action on the heart There is a wide margin between its therapeutic and its toxic doses Convulsions follow overdosage and these are succeeded by depression A drug gradually falling from favour

Used to combat respiratory depression following barbiturates avertin and volatile anaesthetics Supplied in 17 ml and 55 ml ampoules

Given intravenously in 3 to 5 ml doses which can be repeated Additional intramuscular injection prolongs its effect Used in asphyxia neonatorum when it can be injected into the umbilical vein It may do good when infant is depressed due to pre partum narcotic medication of mother

Has been added (1 ml to 1 g) to intravenous barbiturates to prevent respiratory depression which is probably a useless procedure Sometimes used during neurosurgical operations to arouse patients sufficiently for them to co operate in mapping out anaesthetic and hyperaesthetic areas

A dose of 5 ml given rapidly intravenously perhaps preceded by 0.1 g of thiopentone will often cause an explosive cough and aid in clearing the airway in cases of threatened atelectasis Paraldehyde 2 ml intravenously has a similar effect

It has been recommended as a treatment for reversal of apnoea to wake up a muddled respiratory centre (Nosworthy) \* Convulsions are said not to occur when coramine is used under these circumstances

It is converted before excretion into a close derivative of nicotina mide and is related to the antipellagra vitamins

**Picrotoxin** —An old drug the use of which as an analeptic was re introduced in 1931 by Maloney Fitch and Tatum The most potent of this group of analeptic A non alkaloidal extract of the bean *Cocculus indicus* Not used much now

It stimulates the cortex having an antinarcotic action which becomes a convulsant action with toxic doses also stimulates midbrain and the medulla and thus respiration It has no effect on the distribution metabolism or excretion of thiopentone

Used in 0.3 per cent solution the dose being 1-2 ml (3-6 mg) intravenously and repeated after an interval if necessary Muscular twitching is a sign of overdosage It antagonizes effects of the overdosage of morphine avertin ether and barbiturates The greater the depression the less satisfactory is the drug in antidoting it

**Cardiazol (Metrazol Leptazol Phrenazol)**—Pentamethylene tetrazole A camphor derivative used in 10 per cent solution  
A medullary stimulant and in larger doses a convulsant much used in convulsive therapy in psychiatry Unlike coramine has no effect on the carotid and aortic bodies

Average intravenous dose to produce respiratory stimulation 2 ml—carefully repeated if necessary It antagonizes the action of avertin ether barbiturates

**Alpha lobeline**—A pure alkaloid derived from the plant *Lobelia inflata* known as Indian or wild tobacco It stimulates the medulla and the carotid body and lowers the carbon dioxide threshold  
Average dose is 1 ml containing  $\frac{1}{4}$  gr  
Icoral has a similar effect

**Sodium Succinate**—The salt of succinic acid ( $\text{COOH}-\text{CH}_2-\text{CH}_2-\text{COOH}$ )—disodium succinate hexahydrate—30 per cent in distilled water Because of the six molecules of water the actual strength is 18 per cent Stable at room temperature but decomposes on heating

Intravenous injection causes both subjective and objective feeling of warmth without sweating

Initial intravenous dose 5 ml 1 ml per second Up to 20 ml can safely be given and in acute barbituric acid poisoning 200 ml have been given After initial recovery patient may go off to sleep again Not a toxic drug Glucose is not oxidized directly to carbon dioxide and water but is changed by a series of reactions into pyruvates Oxidation of succinate is not inhibited by barbiturates as is glucose lactate and pyruvate Sodium succinate is assumed to provide an oxidation substrate until the barbiturate can be destroyed or excreted

Recent work does not support the claims made for sodium succinate as an analeptic against barbiturates \*

Has been used to counteract thiopentone and morphine depression

**N Allyl Normorphine Hydrobromide (Nalorphine, Nalline, Lethidrone)**—This was first described by McCawley Hart and Marsh in 1940 and synthesized by Weijlard and Erickson in 1942 It was used as an antagonist to morphine by Eckenhoff in 1951 It is closely related chemically to morphine in which there is a nitrogen atom with a methyl group attached in the new drug this methyl group is exchanged for an allyl group ( $\text{C}_3\text{H}_5$ ) It combines with the receptors on which morphine acts (see also annotation in *Brit med J* 1953 1 35 Bodman R 1953 *Proc R Soc Med* 46 11 923 also Landmesser C M Cobb S and Converse J G 1953 *Anesthesiology* 14 535)

It is effective in counteracting respiratory depression and narcosis produced by opiates pethidine and physeptone methorphan and metapon but not that due to ether cyclopropane thiopentone or other barbiturates Intravenous dose 3 mg Stimulation of respiration only lasts 10–15 minutes and may then increase drowsiness and lead to nausea sweating etc Not suitable

**N Allyl Normorphine Hydrobromide continued**

for use in ambulant patients. It restores the EEC pattern of morphinized patients to normal.

It can be used —

- 1 In the treatment of opium poisoning e.g. in neonates in the ill or shocked or emphysematous patients suffering from right heart failure
- 2 In the treatment of morphine addiction
- 3 To aid diagnosis in patients whose symptoms are masked by morphine
- 4 In midwifery to reverse morphine or pethidine induced foetal respiratory depression (Bodman)

Repeated doses may cause hypertension and should not be given unless respiratory depression is severe. It abolishes the increased intestinal tone due to morphine. When given to non morphinized patients it causes nausea, miosis, sweating, slight sedation and hallucinations. It is a tricky drug to use. Too little will not reverse morphine effects, too much will potentiate them. In anaesthesia, respiratory depression due to morphine or pethidine is its chief indication. Useful in the treatment of asphyxia neonatorum due to foetal respiratory depression from opiates or pethidine\*. The dose in the baby is 0.25–0.5 mg given into the umbilical vein and repeated if necessary.

The ordinary solution contains 10 mg per ml. The neonatal solution 1 ml equals 1 mg.

**Levallorphan Tartrate** (Lorfan R 17700) — This is the N allyl derivative of laevo morphinan (laevo dromoran) to which it bears a similar relationship as does nalorphine to morphine. It is 3 hydroxy N allyl morphinan tartrate and is an effective antagonist to the respiratory depression produced by morphine, pethidine, dromoran etc. Without reversing the sensory depressant effects it potentiates the depressant effects of pentobarbitone. In anaesthesia it is used to reverse severe respiratory depression which may accompany nitrous oxide oxygen anaesthesia supplemented with pethidine, alphaprodine etc. Suggested dosage to reverse the respiratory depressant effects of the sedative is one hundredth the pethidine dosage, one twentieth the morphine or alphaprodine dosage and one tenth the levorphan dosage. Put up in 0.1 per cent solution 1 ml equals 1 mg. *Pethilorfan* contains pethidine and levallorphan 100 to 125 (80:1). It is less depressing to the respiratory centre than pethidine without reducing the analgesia of pethidine.

**Methyl Phenidate** (Ritalin) — This is a new analeptic which is stated to aid recovery from anaesthesia. Dose 20 mg intravenously †

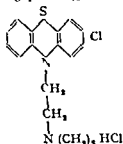
Fckenhoff J. E. Hoffman G. L. and Dripps R. D. *Anaesthesia* 1952 **13** 242  
 † Gale A. S. *Ibid* 1958 **19** 522

## CHAPTER XI

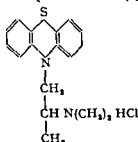
## THE PHENOTHIAZINE DERIVATIVES

This group of drugs includes among hundreds of others —

- 1 **Chlorpromazine Hydrochloride** (3-chloro 10 (3/dimethylamino-n propyl) phenothiazine hydrochloride) (Its synonyms are Largactil Thorazine Megaphen 4560 R P Hibernal)



- 2 **Promethazine Hydrochloride** (N 2 dimethylamino N propyl phenothiazine hydrochloride) (Its synonyms are Phenergan and Atosil) Very useful for premedication (25-50 mg i m)



- 3 **Promethazine 8 chlorothiophyllinate** (Avomine)

- 4 **Diethazine** (Dipacol)

- 5 **Ethopropazine** (Lysivane)

- 6 **Pacatal** (Pecazine Lacumin) ((P 391) 9 (1 methyl 3 piperidyl methyl) Phenothiazine) Compounds (1) (2) and (3) are used in anaesthesia the last as an anti-emetic Compounds (4) and (5) are used in the treatment of Parkinsonism and compound (6) is an ataraxic or tranquillizing drug

The description which follows applies primarily to chlorpromazine

**History**—Chlorpromazine was synthesized in the laboratories in France of Rhône Poulenc Spécia by Charpentier in 1950 the summit of a prolonged research With promethazine it was used



**History continued**

in anaesthesia by Laborit and Huguenard in 1951\* who used the names potentiated anaesthesia lytic cocktail anaesthesia with anaesthetics and by Angus Smith and Fairer in 1953† In the same year Dundee and his colleagues‡ used it together with surface cooling to facilitate hypothermia

**Physical Characteristics**—Chlorpromazine is a pale crystalline powder freely soluble in water Its solutions are acid—pH of 5 per cent solution is 4.5—so it should be well diluted to avoid irritation It is prepared in 0.5 per cent and in 2.5 per cent solution which should not be exposed to light and in tablets each containing 10 or 25 mg Its solution is precipitated when mixed with thiobarbiturates atropin sulphate polyvidone (Plasmo san) and gallamine—the precipitate is however dissolved in excess of gallamine It may cause cloudiness when mixed with certain solutions of dextro e and saline It is miscible with solutions of d-tubocurarine scoline and anectine (but not all brands of suxamethonium) morphine noradrenaline procaine and its amide methamphetamine (methedrine) and of course pethidine and promethazine

**Pharmacology of Chlorpromazine**—It is very variable in its effect especially when taken by mouth Its results begin to show a few minutes after intravenous injection 15–30 minutes after intramuscular injection and 1–1½ hours after being swallowed

- 1 **THE CENTRAL NERVOUS SYSTEM**—It depresses the reticular formations of the brain and has an inhibitory effect on all cellular activity the so called narcobiotic effect (Decourt) It produces drowsiness and relieves anxiety but does not inhibit the higher psychic centres It antagonizes drugs other than strychnine having a stimulating action on the brain stem e.g. nikethemide nicotine and caffeine It gives rise to a bilateral Horner's syndrome The E.E.C. changes are those of normal sleep and differ from those resulting from barbiturates
- 2 **AUTONOMIC NERVOUS SYSTEM**—It inhibits sympathetic activity centrally by depressing the centres in the diencephalon and thus prevents responses to stimuli mediated through sympathetic nerves e.g. vasoconstriction following haemorrhage trauma and shock This central inhibition of vasomotor reflexes is one of the chief characteristics of the drug It may also depress the heat regulating centre in the diencephalon It greatly reduces the effects of adrenaline and in a lesser degree those of noradrenaline and acetylcholine
- 3 **RESPIRATORY SYSTEM**—Pulmonary ventilation may be reduced bronchial and laryngeal reflexes are depressed It may cause Cheyne Stokes respiration Has been used with success in the treatment of asthma Increases respiratory rate depressed by pethidine

Laborit, H. and Huguenard, P. *Pres Med* 1951 59 139

† Smith, A. and Fairer, J. G. *Br J Med* 1953 2 1247

‡ Dundee, J. W. Scott, W. E. B. and Mesham, P. R. *Ibid* 1953 2 1237

- 4 **CARDIOVASCULAR SYSTEM**—Chlorpromazine reduces blood pressure by (a) Causing peripheral vasodilatation the result of central vasomotor depression and (b) Peripherally antagonizing adrenaline and noradrenaline. Thus the reflex vasoconstriction following shock is not seen. The hypotension is potentiated or reduced by posturing the patient. Vasodilatation reduces peripheral resistance and cardiac output is increased. Tachycardia usually accompanies hypotension (Marey's law) but need not be long lasting—it may be due to vagal inhibition. The skin is warm, dry and pale with dilated veins but the capillaries are constricted and this gives a somewhat cadaveric appearance to the patient. Whereas ordinary anaesthesia causes peripheral vasodilatation accompanied by splanchnic vasoconstriction chlorpromazine causes both peripheral and splanchnic vasodilatation. The drug has no marked effect on the E.C.G. and protects against cardiac arrhythmia caused by cyclopropane, trichlorethylene and chloroform. If given before the onset of shock—but not after its development—the drug prevents the intense and prolonged vasoconstriction with its accompanying visceral ischaemia. It reduces the power of adrenaline to delay absorption of local analgesic drugs.
- 5 **VOMITING**—It depresses the chemoreceptor trigger zone of Borison and Wang in the floor of the fourth ventricle and in larger doses depresses the vomiting centre itself. It is a valuable drug in the prevention and treatment of all types of vomiting other than that due to mechanical causes.
- 6 **TEMPERATURE REGULATION**—
- a Depresses the tone of muscle
  - b May depress the heat regulating centre
  - c Causes peripheral vasodilatation so that the patient becomes strongly influenced by the surrounding temperature
  - d Inhibits shivering by a central effect. Oxygen consumption is reduced only if the patient's temperature is made to fall.
- 7 **OTHER ACTIONS**—Chlorpromazine is a local analgesic and also potentiates the local effects of procaine. It potentiates the effects of anaesthetics, analgesics, hypnotics and muscle relaxants. It is not a strong antagonist of histamine nor has it any true ganglionic blocking activity. Noradrenaline and phenylephrine are the preferred drugs should it be desired to reverse the hypotension caused by the drug. The bradycardia usually associated with the noradrenaline drip is not seen if chlorpromazine has been given previously. The secretions of the mouth, pharynx and upper respiratory tract are dried up. It is an antipruritic.
- 8 **SIDE EFFECTS AND TOXIC ACTIONS**—
- a Faintness and dizziness due to postural hypotension
  - b Liver damage following prolonged administration shown by an enlarged tender liver and jaundice of obstructive type. The kidneys are not harmed by the drug.
  - c Contact dermatitis
  - d Agranulocytosis following prolonged administration

**History continued**

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 ‡ Dundee J W Scott, W B. B., and *ibid.*

100-200 mg of pethidine and slowly run until the patient has received 1 ml per lb body weight. Larger amounts may be required.

- b To control hiccough during laparotomy when 5-10 mg are injected intravenously and accompanied by 0.5 mg of phenylephrine intramuscularly to prevent hypotension. The drug can be used for the same purpose in the immediate post-operative period 50 mg being infused as a drip kept up until the patient is comfortable.
- c To reduce labour pains promethazine 25 mg with pethidine 50 mg intramuscularly repeated if necessary.
- d To reduce or cure post-operative vomiting.
- e To aid induced hypothermia by surface cooling.
- f To protect the patient from the serious consequences of overdosage with adrenaline or noradrenaline.
- g In the treatment of tetanus when 25 mg of chlorpromazine and pethidine can be given about four hourly together with nitrous oxide and oxygen actively inspired.\*
- h To aid endoscopy under local analgesia together perhaps with a barbiturate and scopolamine.
- i To control hyperpyrexia.
- j To promote sleep during operations performed under regional analgesia.

Chlorpromazine has been used in general therapeutics for —

- a The potentiation of analgesics in inoperable carcinoma.
- b To cure vomiting due to digitalis radiation carcinomatosis labyrinthitis acute alcoholism anaemia etc.
- c The treatment of eclampsia.
- d In organic psychoses and delirium tremens.

**Pacatal.**—This has been used to relieve anxiety and potentiate anaesthetic before operation in doses e.g. of 100 mg by mouth the night before operation and 150-200 mg intramuscularly 1 to 2 hours before operation. Smaller doses have given disappointing results†. During operation it can be given intramuscularly in doses of 25-100 mg or intravenously in divided doses.

See also the following reviews of phenothiazine drugs —

Viaud, P., *J. Pharm. and Pharmacol.* 1954 6 361.

Dundee, J. W. *Brit. J. Anaesth.* 1954 28 357.

Labont, H. and Huguenard, P. *Pratique de l'Hibernothérapie en Chirurgie et en Médecine* 1954 Paris Masson.

Bodman, R. I., Morton, H. J. V. and Thomas, E. T. *Lancet* 1955 2, 210.

† Flemming Haxholdt B. and Skov Jensen, A. *Acta Anaes. Scand.* 1953 139.

## Pharmacology of Chlorpromazine continued

- 9 **EXCRETION** — Broken down in body perhaps partly by the liver. Small amounts excreted in the urine. Interferes with the iodine Gmelin and Fouchet tests for bile in the urine.

**Promethazine Hydrochloride** — Its effects are similar to those of chlorpromazine but it has among others the following differences

- a It has less antagonism to adrenaline
- b It has 100 times more antagonism to histamine
- c It is a more potent depressant of upper respiratory tract reflexes

## Clinical Uses of Chlorpromazine (and Promethazine) —

## 1 IN ANÆSTHESIA —

- a **AS PREMEDICATION** — Various techniques have been described including the following — Oral administration of 150 mg the night before with 50 mg intramuscularly one hour before operation. 25 mg by mouth the night before the morning before and four hourly after operation for 48 hours. This has a useful anti emetic effect. 200 mg as a rectal suppository two hours pre operatively. 12.5 to 50 mg with an equal amount of promethazine and 50–100 mg of pethidine intramuscularly 1½ hours pre operatively.
- b **AS PART OF THE ANÆSTHETIC TECHNIQUE** — Again various methods of administration have been advocated including (i) The slow intravenous drip of 500 ml of dextrose containing e.g. chlorpromazine 50–100 mg, promethazine 50–100 mg and pethidine 50–100 mg. The drip can be slowed down when the desired result has been obtained. (ii) The same amount of the three drugs can be dissolved in 20 ml of dextrose solution and given slowly intravenously or intramuscularly before anæsthesia commences. The patient is likely to be sleepy for 5–10 hours after the administration of these amounts and of course general anæsthetic agents will be used in smaller doses than normal.

The writer has found the drip technique very useful in such operations as thyroidectomy, radical mastectomy, mastoidectomy, fenestration etc. \* and intra ocular operations. Hypotension can be increased by tilting the head upwards and decreased by tilting it downwards together with the intravenous injection of noradrenaline or phenylephrine 0.5 mg. For these and other operations he prefers to use in addition small doses of thiopentone and relaxant for intubation and gas and oxygen for maintenance. Straining and coughing are minimized and the long post operative sleep may in such cases be beneficial. Chlorpromazine is more depressant than promethazine and its dose should usually be kept small.

Other anæsthetic uses include —

- a To produce sedation during cardiac catheterization in children a drip is set up containing 50 mg of chlorpromazine and

100-200 mg of pethidine and slowly run until the patient has received 1 ml per lb body weight. Larger amounts may be required.

- b To control hiccough during laparotomy when 5-10 mg are injected intravenously and accompanied by 0.5 mg of phenylephrine intramuscularly to prevent hypotension. The drug can be used for the same purpose in the immediate post-operative period 50 mg being infused as a drip kept up until the patient is comfortable.
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## CHAPTER XVI

## SPINAL ANALGESIA

## INTRADURAL SPINAL ANALGESIA

**History** — Cerebrospinal fluid discovered by Cotugno in 1764 its circulation described by Magendie in 1825

Cocaine isolated from *Erythroxylon coca* in 1859 by Niemann and Lossen its analgesic properties described by Schraff in 1862 and V Ansep in 1880 Introduced into medicine as local analgesic for ophthalmology by Karl Koller and Sigmund Freud in 1884

First spinal analgesia by J Leonard Corning New York neurologist in 1885 He accidentally pierced the dura while experimenting with cocaine on the spinal nerves of a dog Later he deliberately repeated the intradural injection called it spinal anaesthesia and suggested it might be used in surgery Be the destiny of this observation what it may it has seemed to me on the whole worth recording (Corning)

Lumbar puncture standardized as a simple clinical procedure by Quincke in 1891 in Germany and by Essex Wynter in England in the same year

First planned spinal analgesia for surgery in man performed by August Bier of Kiel in 1898 using 2 c.c. of 1 per cent cocaine Advised it for operations on legs but gave it up owing to toxicity of cocaine Tuffier and Sicard soon afterwards extended its scope to include the external genitals Tait and Cagliari and also Rudolf Matas were its first users in the US in 1899 their works being published in the following year

Stovaine synthesized by Fournieu (Fr *fournieu stove*) in 1904 and novocain (procaine) described by Einhorn the same year

Barker of London was the first to realize importance of curves of vertebral canal and the use of gravity in control of level of analgesia He and Chaput independently introduced heavy stovaine solutions in 1907 Babcock first to use light solution his formula containing stovaine alcohol lactic acid strychnine etc

Spinal analgesia little used until Caston Labat's work in 1921 He urged use of neocaine (procaine) crystals dissolved in cerebrospinal fluid together with barbotage and early Trendelenburg position Then came George Pitkin with his light (spinocain) and heavy (duracaine) solutions and his use of the fine bore short bevel needle (1927)

Chen and Schmidt introduced ephedrine in 1923 while Ocherblad and Dillon used it to maintain the blood pressure in spinal analgesia in 1927

Meischer discovered the analgesic properties of percaine (nupercaine) in 1929 while Howard Jones published his tech. — 1930

Etherington Wilson's work appeared in 1933 and Walter Lemmon's first account of continuous spinal analgesia was published in 1940

Since in the U.S. popularized amethocaine (tetracaine) which was synthesized by Lissle in 1928

In most parts of the U.K. intradural spinal analgesia is at present under a cloud partly because of the tendency to litigation should complications follow \* such as the Woolley and Roe case. Anxiety is made worse as the cause of some of these complications such as adhesive arachnoiditis are not fully understood. The method is however a valuable one and if proper care is taken in the technique it yields in a very large proportion of cases admirable results and satisfaction to all concerned. Extradural analgesia can be substituted for spinal analgesia in nearly every case

### ANATOMY

**The Vertebrae**—The vertebral column consists of seven cervical twelve thoracic five lumbar five sacral and four or five coccygeal vertebrae. The sacral and coccygeal vertebrae are fused in adult life

A typical *lumbar vertebra* consists of —

- 1 A body wider from side to side than from before backwards and kidney shaped
  - 2 Two pedicles strong and directed backwards from the upper part of the body. Each pedicle is notched more inferiorly than superiorly and through the intravertebral foramina formed by the two notches of contiguous vertebrae the spinal nerves emerge. The intravertebral foramina may be accidentally entered and the dura perforated by a needle during the induction of paravertebral analgesia.
  - 3 Two laminae meeting posteriorly and enclosing the vertebral foramen which is triangular and larger than in the thoracic but smaller than in the cervical region
  - 4 The spinous process thick broad and quadrilateral. It rises from the point of union of the laminae projects backwards and ends in a rough uneven border
  - 5 Two superior and two inferior articular processes project respectively upwards and downwards from the junctions of the pedicles and laminae. In the articulated column the inferior articular processes are embraced by the superior articular processes of the adjacent vertebra
  - 6 Two transverse processes homologous with the ribs situated in front of the articular processes and rising from the junction of the pedicles and laminae in the case of the upper three and from the pedicles in the lower two
  - 7 The mammillary and accessory processes on each side
- Whereas the thoracic vertebrae have articular facets for the ribs the lumbar vertebrae have not

## Anatomy continued

**The Inter-vertebral Fibrocartilages**—These are interposed between adjacent surfaces of the vertebral bodies. They give the vertebral column its flexibility and constitute about one-quarter the length of the column. Each is composed of—

- 1 The nucleus pulposus in the centre the remains of notochord. A jelly like mass existing under considerable tension.
- 2 The annulus fibrosus concentric and radial fibres attached to the nucleus centrally.
- 3 The cartilage plates above and below the nucleus.

Nuclear retropulsion may follow a clumsy lumbar puncture. The symptoms are those of sciatica.

Infected intervertebral disk has also been reported. It leads to sinus formation.

**The Vertebral Column.**—This has four curves of which the thoracic and sacral are primary and are concave anteriorly. The cervical and lumbar curves are secondary and are convex anteriorly. The cervical curve is developed when the baby first holds up its head.

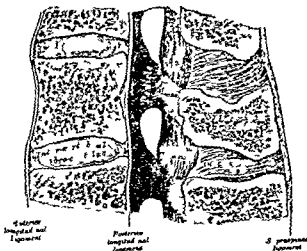


Fig 49.—Median sagittal section of two lumbar vertebrae and their ligaments. (From Gray's Anatomy by kind permission of P. Fisher, T. B. Johnston.)

the lumbar when it begins to walk. The degree of curvature varies in different individuals and is modified by posture. Thus when the spine is fully flexed, the cervical and lumbar curves are obliterated. In the supine position the third lumbar vertebra marks the highest point of the lumbar curve while the fifth thoracic is the lowest point of the dorsal curve. Kyphosis, lordosis, scoliosis and hypertrophic arthritis of the spine may upset the curves and make lumbar puncture difficult.

The direction of the spinous processes determines the direction in which the spinal needle must be inserted. The spinous processes of the cervical the first two thoracic and the last four lumbar vertebrae are all practically horizontal and are therefore opposite the bodies of their respective vertebrae. The other spinous processes are inclined downwards their tips being opposite the bodies of the vertebrae next below except the tip of the first lumbar is opposite the intervertebral disk (Fig 49)

**The Vertebral Canal**—Bounded in front by bodies of the vertebrae and intervertebral disks posteriorly by arch which bears spinous processes and by ligaments between them called the interspace laterally by pedicles and laminae. Size and shape varies but is larger in cervical and lumbar regions

#### CONTENTS—

- 1 Roots of spinal nerves
- 2 Spinal membranes with their enclosed spinal cord and cerebro spinal fluid
- 3 Structures—vessels fat and areolar tissue of extradural space

**The Vertebral Ligaments bounding the Canal—**

- 1 SUPRASPINAL LIGAMENT passes longitudinally over tips of spinous processes
- 2 INTERSPINAL LIGAMENTS joining spinous processes together
- 3 LIGAMENTA FLAVA running from lamina to lamina composed of yellow elastic fibres. Half of the substance of the posterior wall of the vertebral canal is composed of the bony laminae half by the ligamenta flava
- 4 POSTERIOR LONGITUDINAL LIGAMENT within the vertebral canal on posterior surfaces of bodies of vertebrae from which it is separated by the basivertebral veins

Midline spinal puncture pierces the first three of these. In lateral approach only ligamenta flava are encountered. Bleeding may result from puncture of basivertebral veins

**The Spinal Cord**—The elongated part of the central nervous system which occupies upper two thirds of vertebral canal. Extent is from upper border of atlas to upper border of second lumbar vertebra and lower still in infants. Above continuous with brain below ends in conus medullaris from apex of which filum terminale descends as far as coccyx. In foetal life length of cord corresponds with that of vertebral canal but the canal grows more rapidly than the cord. Thus nerve roots which pass out transversely in early foetal life come to be more and more oblique in direction so that in adult life lumbar and sacral nerves descend almost vertically to meet their foramina and are known as the cauda equina

In cervical region a vertebral spine is one lower in number than corresponding cord segment

In upper thoracic region a vertebral spine is two lower in number than corresponding cord segment

In lower thoracic region spine is three lower than cord segment so tenth spine is opposite first lumbar segment

**Anatomy—The Spinal Cord** *continued*

Spinal puncture above the L 2-3 interspace may result in cord injury but elements of cauda equina although they may be touched by the point of the needle are not easily damaged seriously

The spinal cord is ensheathed by three membranes from without inwards —

**DURA MATER**—A strong fibrous layer forming a tubular sheath attached above to margins of foramen magnum and ending below at lower border of second sacral vertebra. Separated from bony wall of vertebral canal by the epidural space which contains fats areolar tissue and a venous plexus and the anterior and posterior roots of the spinal nerves. Its main fibres are longitudinal so the lumbar puncture needle should be introduced with its bevel separating rather than dividing these fibres. Below the dural sac ends at the level of the second sacral vertebra.

**ARACHNOID**—This is a thin transparent sheath closely applied to the dura the subdural space being merely a capillary layer.

**PIA MATER**—This is separated from the arachnoid by the sub-arachnoid space filled with cerebrospinal fluid. Here local analgesic drugs are deposited in spinal analgesia. The pia closely invests the cord and sends delicate septa into its substance. From each lateral surface of the pia mater a fibrous band the denticulate ligament projects into the subarachnoid space and is attached by a series of pointed processes to the dura. The pia mater ends as a prolongation—the filum terminale—which pierces the distal end of the dural sac and is attached to the periosteum of the coccyx.

There are two enlargements of the cord one in the cervical the other in the lumbar region corresponding to the origins of the nerves of the arms and legs.

**Spinal Segments**—The cord is divided into segments by the pairs of spinal nerves which arise from it. These pairs are thirty-one in number and are as follows (a) Eight cervical (b) Twelve thoracic (c) Five lumbar (d) Five sacral (e) One coccygeal.

The nerve roots within the dura have no epineurial sheaths and are therefore easily affected by doses of analgesic drugs brought into contact with them. The cord is not transversely blocked by spinal analgesia but it is probable that there may be some block of the longitudinal columns by penetration of the drug.

**Spinal Nerves**—Thirty-one pairs each arising from the cord by two groups of fibres called anterior and posterior roots.

*Anterior root* is efferent and contains fibres subserving —

- 1 Motor to voluntary muscles
- 2 Preganglionic fibres to sympathetic chain in the region from thoracic 1 to lumbar 3 inclusive these later become the white rami communicantes and carry constrictor impulses to vessels ducts and bronchi and dilator impulses to vessels (sacral 2 3 and 4 also have white rami accompanying them the nervi erigentes)

*Posterior root* is larger than anterior. All the afferent impulses from the whole body including viscera pass into the posterior roots.

Each posterior root has a ganglion and conveys fibres of —

- |  |   |
|--|---|
| 1 Pain   | } These may be coarse (protopathic) or fine (epicritic) |
| 2 Tactile  |   |
| 3 Thermal sensation                                      |   |
| 4 Deep or muscle sensation from bones joints tendons etc |   |
| 5 Afferents from the viscera (sympathetic)               |   |
| 6 Vasodilator fibres                                     |   |

Pain and temperature fibres entering the posterior horn where they end round cells in the grey matter. Fibres then cross to the contralateral side within three segments and ascend in the lateral spinothalamic tract to the thalamus.

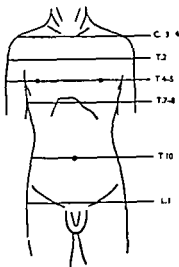


Fig. 50—Segmental levels.

Tactile impulses ascend in the ventral spinothalamic tract to the thalamus.

Deep or muscle sensory impulses ascend in the posterior columns and spinocerebellar tracts.

Vibration impulses ascend in the posterior columns.

The anterior and posterior roots unite in the intervertebral foramina to form the main spinal nerve-trunks which soon divide into anterior and posterior primary divisions—mixed nerves. These are blocked only secondarily in spinal analgesia. It is block of the nerve roots which gives the effect. There is evidence however that analgesic drugs after subarachnoid injection can soak along the nerve trunk for as much as 2 cm beyond the intervertebral foramen. Analgesic drugs affect autonomic, sensory, and motor fibres in that order while fibres which block easily hold the drug.

**Anatomy—Spinal Nerves** *continued*

longest, thus sensory block lasts longer than motor and usually ascends two segments higher up the cord than motor block

**SEGMENTAL LEVELS** (*Fig 50*)—Perineum S 1-4 Inguinal region I 1 Umbilicus T 10 Subcostal arch T 6-T 8 Nipple line T 4 5 Second intercostal space T 2 Clavicle C 3-4

The skin above the nipple line has a double innervation from C 3 and C 4 and from T 2 T 3 and T 4 so even with a successful block to C 8 there will be some sensation above the nipple line The success of a block to T 1 is proved by the inability of the patient to hold a sheet of paper between the fingers (innervation of interossei C 8 and T 1)

**SEGMENTAL LEVELS OF SPINAL REFLEXES**—

Epigastric T 7 8	Plantar S 1 2
Abdominal T 9 12	Knee jerk L 2-4
Cremasteric L 1 2	Ankle jerk S 1 2

**The Subarachnoid Space**—This is between the arachnoid and the pia mater and in the lumbar region it occupies more than half the anterior posterior diameter of the vertebral canal Communicates with the ventricular system of brain by —

- 1 Foramen of Magendie a median opening in the roof of the fourth ventricle
- 2 The foramina of Luschka two small passages in the lateral recesses of the fourth ventricle
- 3 The foramina of Elze and Retzius in the roof of the fourth ventricle

The contents of the space are the spinal nerve roots the denticulate ligaments a spongy reticulum of fibres connecting the pia to the arachnoid the cerebrospinal fluid.

**Circulation of Cerebrospinal Fluid**—Called by Harvey Cushing the third circulation It forms a short circuit between the arterial and venous circulations

Fluid which is formed by the choroid plexuses in the lateral ventricles passes through on each side the foramen of Monro to join that formed by choroid plexuses in the third ventricle thence through aqueduct of Sylvius to fourth ventricle Fluid leaves this for the subarachnoid space through the central foramen of Magendie and the lateral foramina of Luschka Key and Retzius and reaches the cisterna magna It bathes the whole of the central nervous system and is absorbed into the venous sinuses through the arachnoidal villi This circulation takes no part in spinal analgesia

Ordinary doses of analgesic drugs injected into the spinal subarachnoid space do not reach the fourth ventricle which contains in its floor the centres for the heart and for respiration

**The Cerebrospinal Fluid**—

**SOURCE**—From the choroid plexuses of the third fourth and lateral ventricles by either secretion or dialysis

**REMOVAL**—Into the venous sinuses of the brain via the arachnoidal villi and into the lymph stream via the Pacchionian bodies.

Movement of the fluid is slow and of no importance in spinal analgesia. When injected into the subarachnoid space drugs such as novocain are absorbed into the blood stream.

**PHYSICAL CHARACTERISTICS**—Clear and colourless with slight opalescence due to globulin. Sp. g. at 37°C is  $10010 \pm 0.0003$  g per ml. The density is more dependent on the temperature and on the contained sodium chloride and carbon dioxide than on the contained protein. Increased in diabetes, uræmia and old age. Poor in cellular elements, five or less per c mm (lymphocytes); an increase of cells indicates meningeal irritation. Quantity 110–150 ml. Volume of spinal-cerebrospinal fluid about 25 ml, 15 ml of which is below T5. Pressure 70–180 mm of water in the lateral level position, 375–550 mm of water in vertical position. Owing to hydrostatic pressure of column of fluid, cerebrospinal fluid drips faster from a needle in a sitting patient than from one in a lateral patient. Pressure influenced directly by intracranial venous pressure and by total amount of body fluid. Pressure increased in sleep, uræmia, alcoholism and cerebral neoplasm, also in cases of raised  $\text{CO}_2$  tension, congestive heart failure and mediastinal tumour. Factors raising venous pressure such as straining, coughing etc. will raise the cerebrospinal fluid pressure. Decreased in wasting diseases, traumatic leakage of cerebrospinal fluid and under the influence of large narcotic doses of barbiturates. Pressure controlled by central hypothalamic mechanism. About 500 ml can be secreted in 24 hours if there is a free leak from the subarachnoid space. Changes in the osmotic pressure of the blood affect cerebrospinal fluid pressure, e.g. intravenous injection of hypertonic saline or glucose lowers cerebrospinal fluid pressure.

**CHEMICAL CHARACTERISTICS**—Alkaline, pH 7.6. Protein low, 40 mg per 100 ml. Sugar 45–80 mg per cent. Sodium chloride 0.75 per cent. Antibodies etc. not found in cerebrospinal fluid, hence great risks of infection. Drugs are not secreted into it except urotropine and sulphonamides. After spinal analgesia both albumin and globulin increase. Alkalinity and sugar content lower, magnesium content higher than in blood. Contains small amounts of cholinesterase but not sufficient to inactivate such local analgesics as procaine, amethocaine or lignocaine.

#### **FUNCTIONS**—

1. It is a fluid cushion to protect the brain and spinal cord from trauma.
2. By its absorption and formation according to need it regulates the volume of the cranial contents.
3. It has a slight function in the metabolic exchanges of nervous tissue and may take the place of lymph. Intravenous injection of large amounts of normal saline or distilled water cause a rise in pressure. Intravenous injection of hypertonic solutions, e.g. 50 ml of 10 per cent saline, 100 ml of 25 per cent glucose or 100 ml of 50 per cent sucrose cause a lowering of pressure.



**Anatomy—Cerebrospinal Fluid** *continued*

Non inflammatory overproduction—liquorrhœa—is seen in cases of nasal sinus infection in otitis at high altitudes (hypoxia) and after concussion and lumbar puncture

The opposite condition is aliquorrhœa and may occur spontaneously in dehydration or after lumbar puncture

**FACTORS OF SPECIAL SIGNIFICANCE**

**Spinal Analgesia and Respiration.**—The respiratory centre in the floor of the fourth ventricle depends for its action on the amounts of oxygen and carbon dioxide in the blood. Paralysis of respiratory muscles may lead to anoxia of the centre with further depression. Hence oxygen administration during spinal analgesia is a rational procedure. (Although recent work\* fails to demonstrate the existence of hypoxia and hypercarbia with spinal analgesia.)

The phrenic nerve supplying the diaphragm rises from the anterior roots of C 3, 4 and 5 and should not be encroached on in spinal analgesia.

Intercostal muscles supplied by nerves T 1–12 thus any motor block encroaching further than T 12 by paralysing intercostal muscles will depress respiration in proportion to height of block. *Note* muscular block is usually one or two segments below sensory.

**Spinal Analgesia and Heart rate** —The cardiac rate is accelerated by stimulation of the cardiac sympathetic nerves. White rami leave the cord with the upper four or five thoracic nerves run up the sympathetic chain to the cervical region and then as post ganglionic fibres after synapsing reach the myocardium as cervical cardiac nerves. Three of these on each side one from upper middle and lower cervical sympathetic ganglia. Other fibres run to corresponding ganglia of sympathetic chain and from there pass as post ganglionic fibres to the cardiac plexus (thoracic cardiac nerves). Slowing of the heart rate is caused if any of the anterior roots carrying these fibres is blocked as may happen in high spinals. A further cause of slow pulse rate is the lowering of blood pressure in the right auricle consequent on diminished venous return (Bainbridge effect). On the other hand tachycardia during spinal analgesia may result from the operation of Marey's law inversely correlating heart rate and systolic blood pressure. Bradycardia is the more frequent effect.

**Physical Factors** —Analgesic solutions injected into the subarachnoid space are influenced by —

- a Dispersion i.e. mechanical mixing from force of injection
- b Convection by gravity e.g. heavy nupercaine
- c Displacement of cerebrospinal fluid by large volumes of fluid (as with hypobaric nupercaine)
- d Diffusion gradual intermingling by osmotic tension of no practical importance

**Factors influencing Height of Analgesia —**

- 1 **POSITION OF PATIENT DURING INJECTION** — Sitting hyperbaric solutions tend to fall and hypobaric solutions to rise. Lateral position unilateral analgesia is more pronounced than bilateral if injections are made slowly and patient remains on side during fixation of the drug. Gradual spread to the other side usually results.
- 2 **INTERSPACE CHOSEN** — The higher the interspace chosen for injection the higher will be the resulting analgesia leaving other factors out of account. A good rule is to choose L 2-3 interspace for upper abdominal cases. L 3-4 interspace for lower abdominal and leg operations and the L 4-5 interspace for perineal procedures. In the very tall a space higher is sometimes advantageous while in the very short one space lower may be chosen.
- 3 **VOLUME OF FLUID INJECTED** — Height of analgesia is directly proportional to amount of fluid injected.
- 4 **BARBOTAGE** (Fr *barboter* to dabble to paddle to mix a name given to the method by La Fliatre in 1920) — A method of mixing the solution with a greater volume of cerebrospinal fluid than the syringe will hold and of increasing dispersion. It decreases the concentration and sp g of the solution and so lessens the effects of gravity after injection. E.g. if syringe contains 2 ml withdraw 1 ml and inject  $1\frac{1}{2}$  ml withdraw 1 ml and inject  $1\frac{1}{2}$  ml withdraw 1 ml and inject  $1\frac{1}{2}$  ml etc.
- 5 **DOSE OF DRUG INJECTED** — Nerve tissue absorbs local analgesic drugs like blotting paper absorbs ink. A limited amount of nerve tissue can only absorb so much drug the surplus being available for convection or absorption into the blood stream. The greater the concentration the longer will its effect last.
- 6 **FORCE AND RATE OF INJECTION** — Height is directly proportional to this. A slow gentle injection is necessary to get full benefit of sp g differences as in unilateral blocks.
- 7 **SPECIFIC GRAVITY OF SOLUTION** — The sp g of injected solutions should always be known and they should be classed as hyperbaric hypobaric or isobaric. In the case of the first two subsequent posture has a great influence on the level of analgesia. It requires but a few points difference for the effect of sp g to be shown. It is impossible to add any soluble solid to cerebrospinal fluid without raising its sp g.
- 8 **POSTURE OF PATIENT AFTER INJECTION** —
  - If patient remains on the side curves of spine are without effect and gravity of solution controls side of analgesia.
  - If patient is supine —
    - a Hyperbaric solutions pass to the bottom of sacral and dorsal curves some of it to both if injection is made at apex of lumbar curve. Lowest point of dorsal nerve coincides with the 6th thoracic vertebra so upward spread of a reasonable volume of a heavy solution will not occur unless a steep Trendelenburg position is assumed. Similarly hyperbaric analgesic fluid placed in the sacral curve cannot spread upwards over the hollow of the back without a head down

**Factors influencing Height of Analgesia** *continued*

tilt Thus for blocks above the perineum with these solutions the head of the patient should be slightly inclined downwards during injection

- b Hypobaric solutions gravitate to the top of the lumbar curve but few solutions are truly hypobaric
- c Isobaric solutions are uninfluenced by gravity and their maximum effect is at point of injection

In the lithotomy position lumbar curve is obliterated In Trendelenburg's position hypobaric solutions travel caudad while hyperbaric solutions move cephalad The normal spinal curvature limits this movement Raising the head and shoulders accentuates the dorsal curve and tends to prevent spread of hyperbaric solutions to the cervical area with its phrenic roots In Fowler's position the opposite is true

As an analgesic solution travels it leaves some of its drug behind fixed to nerve tissue and thus gradually becomes more dilute This is the so called brake action

**Duration of Analgesia** —Depends on the drug used The upper end of an abdominal incision regains sensation before the lower end Nupercaine and amethocaine last longer than lignocaine and procaine

**Fixation Time** —For lignocaine about five minutes For novocain 3-15 minutes Amethocaine and nupercaine take longer These drug are eventually absorbed into the blood stream ascend via the azygos vein and are destroyed in the liver

**DRUGS USED TO PRODUCE SPINAL ANALGESIA**

(For Pharmacology see Chapter XVIII)

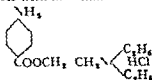
**Cocaine** —This was the first drug used followed by tropacocaine stovaine novocain nupercaine amethocaine and metycaine lignocaine and hexylcaine

Novocain nupercaine amethocaine and metycaine are the chief drugs used to day Cocaine is now given up entirely

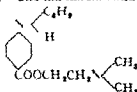
**Stovaine (Amylocaine BP)** —Was popular for many years but is now known to be irritating and has lost much of its popularity It is put up in 2 ml ampoules containing 5 per cent stovaine in 5 per cent glucose sp g 1025 dose 1-2 ml this is Barker's formula Chaput's solution is supplied in 1 ml ampoules containing 10 per cent stovaine in 10 per cent sodium chloride sp g 1080 the dose being 0.6-1 ml Contains no para aminobenzoic radical

**Procaine (Ethocaine BP Novocain Neocaine Syncaine Scurocaine Planocaine Kerocain etc)** —Synthesized by Einhorn in 1904 and advocated by Braun Put up in ampoule containing dry crystals in amounts varying from 50 to 300 mg and in solutions of 5 per cent and 10 per cent The crystals are dissolved in cerebrospinal fluid and injected while the 10 per cent solution is usually diluted with an equal volume of cerebrospinal fluid before injection In 5 per cent strength or less novocain is not irritating to nervous tissue and meninges Spinocain is Pitkin's

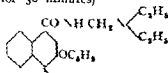
solution and contains novocain 0.195 g (10 per cent) strychnine sulph 0.0022 g starch paste 0.13 g alcohol 0.374 g normal saline to 2.2 ml The sp g is 1.0005 Duracaine is 10 per cent procaine benzoate solution with sp g 1.00 Analgesia lasts from forty to eighty minutes with novocain



**Amethocaine Hydrochloride B.P. (Tetracaine U.S.P. Anethaine Decicain Pantocaine Pontocaine Butethanol)**—Was synthesized in 1908 by O. Eisleb. Can be autoclaved or boiled on one or two occasions. Of slower onset but of longer duration than novocain lasting  $1\frac{1}{2}$ – $2\frac{1}{2}$  hours. Put up in ampoules containing 20 mg of dried powder also in solution 1 per cent each ml containing 10 mg. Spinal D is a 0.5 per cent solution in 6 per cent glucose with sp g 1.025 spinal D isotonic is 0.4 per cent amethocaine in 4.6 per cent glucose and distilled water to make up 5 ml sp g 1.018 and pH 5. The maximum intrathecal dose is 70 mg.



**Nupercaine (Percaine Cinchocain B.P. Dibucaine)**—This is 2-butoxycinchoninic acid 2-diethylamino ethylamide hydrochloride. Introduced in 1909 by Meischer, it is not a cocaine derivative but it is allied to quinine. It too has a slower onset but more lasting effect than novocain and may give analgesia for  $1\frac{1}{2}$ –3 hours. Like amethocaine it is easily destroyed by traces of alkali so that needles, syringes, etc. should be washed through with nupercaine solution which is subsequently discarded. Alkali causes visible precipitation and cloudiness which disappears on the addition of weak acid. Weight for weight it is highly toxic but not dose for dose. Can be boiled or autoclaved without decomposition on one or two occasions (autoclaved at 115°C at 10 lb pressure for 30 minutes).



Ampoules supplied in the U.K. are of three types—

- The 20 ml ampoule of hypobaric solution 1–1500 nupercaine in 0.5 per cent saline with sp g 1.0036 at 37°C. Each ampoule contains 13.3 mg nupercaine.

**Factors Influencing Height of Analgesia** *continued*

tilt. Thus for blocks above the perineum with these solutions the head of the patient should be slightly inclined downwards during injection.

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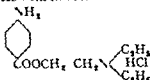
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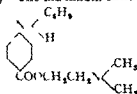
**Stovaine (Amylocaine B.P.)** — Was popular for many years but is now known to be irritating and has lost much of its popularity. It is put up in 2 ml ampoules containing 5 per cent stovaine in 5 per cent glucose, sp. g. 1.025, dose 1–2 ml. This is Barker's formula. Chaput's solution is supplied in 1 ml ampoules containing 10 per cent stovaine in 10 per cent sodium chloride, sp. g. 1.080, the dose being 0.6–1 ml. Contains no para-aminobenzoic radical.

**Procaine (Ethocaine B.P.)** Novocain, Neocaine, Syncaine, Scurocaine, Planocaine, Kerocain, etc. — Synthesized by Einhorn in 1904 and advocated by Braun. Put up in ampoules containing dry crystals in amounts varying from 50 to 300 mg. and in solutions of 5 per cent and 10 per cent. The crystals are dissolved in cerebrospinal fluid and injected while the 10 per cent solution is usually diluted with an equal volume of cerebrospinal fluid before injection. In 5 per cent strength or less novocain is not irritating to nervous tissue and meninges. Spinocain is Pitkin's

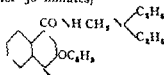
solution and contains novocain 0.195 g (10 per cent) strychnine sulph 0.0022 g starch paste 0.13 g alcohol 0.324 g normal saline to 2 ml The sp g is 1.0005 Duracaine is 10 per cent procaine benzoate solution with sp g 100. Analgesia lasts from forty to eighty minutes with novocain



**Amethocaine Hydrochloride BP** (Tetracaine USP Anethaine Decicain Pantocaine Pontocaine Butethanol) — Was synthesized in 1928 by O. Eisleb. Can be autoclaved or boiled on one or two occasions. Of slower onset but of longer duration than novocain lasting 1½–2½ hours. Put up in ampoules containing 20 mg of dried powder also in solution 1 per cent each ml containing 10 mg. Spinal D is a 0.5 per cent solution in 6 per cent glucose with sp g 1.025. Spinal D isotonic is 0.4 per cent amethocaine in 4.6 per cent glucose and distilled water to make up 5 ml sp g 1.018 and pH 5. The maximum intrathecal dose is 0 mg.



**Nupercaine** (Percaine Cinchocain BP Dibucaine) — This is 2-butoxycinchoninic acid 2-diethylamino ethylamide hydrochloride. Introduced in 1909 by Meischer is not a cocaine derivative but it is allied to quinoline. It too has a slower onset but more lasting effect than novocain and may give analgesia for 1½–3 hours. Like amethocaine it is easily destroyed by traces of alkali so that needles syringes etc. should be washed through with nupercaine solution which is subsequently discarded. Alkali causes visible precipitation and cloudiness which disappears on the addition of weak acid. Weight for weight it is highly toxic but not dose for dose. Can be boiled or autoclaved without decomposition on one or two occasions (autoclaved at 115°C at 10 lb pressure for 30 minutes).



Ampoules supplied in the U.K. are of three types —

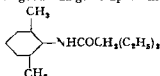
- The 10-ml ampoule of hypobaric solution 1–1500 nupercaine in 0.5 per cent saline with sp g 1.0036 at 37°C. Each ampoule contains 13.3 mg nupercaine.

**Nupercaine continued**

- b The 3 ml ampoule containing 1-200 nupercaine with 6 per cent glucose sp g 1024 at 37 C hyperbaric solution (Silverton 1934) Each ampoule contains 15 mg of nupercaine
- c 2 ml and 3 ml ampoules of isobaric nupercaine 1-200 in buffered solution with sp g 1006 (Keyes and McLellan 1930)

**Metycaine (Neothestin)**—Synthesized by McElvain of the University of Wisconsin (1927) Not a derivative of *p* amino benzoic acid and so can be used without inactivating sulphonamides. A little stronger and lasts a little longer than an equal weight of novocain. Used as 2 ml ampoules of 10 per cent solution usually diluted with equal volume of cerebrospinal fluid. The specific gravity of 5 per cent in Ringer's solution is 1.0046 and of 1.5 per cent in Ringer's solution 1.0023 at 37 C.

**Lignocaine (Xylocaine Lidocaine)**—This is *N*-diethylamino 2,6-dimethylacetanilide and was synthesized by Lofgren and Lundquist in 1946. It is non-irritating, is stable to heat and relatively non-toxic. For spinal analgesia has been used in a strength of 5 per cent with dextrose 3.1 per cent, the specific gravity being 1.018 and the pH 6.5. 1.5 ml of this solution gives about 2 hours of good analgesia up to the umbilicus\*.



**Cyclaine (Hexylcaine hydrochloride)**†—This drug is 1-cyclohexyl amino 2-propyl benzoate hydrochloride. It was synthesized by Cope and Hancock in 1944. It is soluble in water and not decomposed by boiling or autoclaving. More toxic than procaine, less so than metycaine. Duration of anaesthesia 1-1½ hours or with 0.5 mg of adrenaline (0.5 ml 1-1000 solution) 2½ hours. Put up in 2 ml ampoules and each containing 50 mg in 10 per cent glucose. Synthesized in 1944. A good topical analgesic in 5 per cent solution, while 2 per cent solution is used for extradural block and 1 per cent for infiltration.

## AGENTS USED TO RAISE THE BLOOD PRESSURE IN SPINAL ANALGESIA

Pressor drugs act (1) Peripherally ( ) Centrally on the heart. Factors to be considered: Mode of action on vessels or heart; duration of pressor effect; effectiveness of repeated doses; presence or absence of side-effects. Drugs of this nature should not be used routinely but only when a fall of blood pressure is anticipated or actually occurs.

Blood pressure is maintained by impulses passing from the vasomotor centre in the medulla via the anterior spinal roots and white rami from

Adams, B. W., *Anaesthesia*, 1956 11 297

† Anderson, E., and Rubin, J. E., *Anesthesiology* 1952 13 July 429.

T<sub>1</sub> to L<sub>3</sub> and the grey rami to the muscular tissue in the walls of the vessels. In spinal intradural and extradural analgesia these impulses are blocked by paralysis of the pre ganglionic fibres in the anterior nerve roots.

Any drug raising the blood pressure removes one of the great advantages of spinal analgesia—it spoils the bloodless field.

**Ephedrine**—Introduced into Western medicine in 1923 by Schmidt and Chen. The active principle (isolated in 1885 by Yamanashi) of ma huang a Chinese plant. Chemically allied to adrenaline. Its pharmacological action is to inhibit amine oxidase, a tissue enzyme which destroys adrenaline (Gaddum and Kwiatkowski 1938) and this action is akin to that of neostigmine which inhibits cholinesterase—one protects and spares adrenaline the other acetylcholine. It does not act directly on the effector cells but is a potent sympathetic stimulant raising blood pressure stimulating the myocardium constricting arterioles relaxing smooth bronchial and intestinal muscle dilating the pupil and stimulating the cortex and medulla. Probably dilates the coronary arteries and overcomes the coronary constriction caused by pituitary extract. Its vasopressor effects are not reversed by adrenergic blocking agents—unlike those of adrenaline. It dilates the vessels of skeletal muscles. It is not oxidized by amine or phenol oxidase but is excreted unchanged by the kidneys completely in 24 hours. Used in states of hypotension asthma heart block urticaria narcolepsy and enuresis. The larvo-rotatory form is the more active. It thus delays the rate of destruction of adrenaline and so maintains a high blood adrenaline level. Its direct vasoconstrictive action on the vessels is a secondary effect. The rate and also the force of cardiac contraction are increased. Duration of effect is prolonged from 30 to 40 minutes after intramuscular injection but repeated doses not as effective as initial dose (tachyphylaxis). Causes subjective feeling of apprehension trembling and discomfort. It increases the tone of the bladder sphincter and this may be one cause of post-operative retention. It may tend to produce wakefulness either during or after operation—an action shared by methylamphetamine. It has a local analgesic effect. Put up in ampoules of  $\frac{1}{2}$  gr. Dose  $\frac{1}{2}$  to  $1\frac{1}{2}$  gr (30–100 mg).

**Methoxamine** (Vasoxine Vasoxyl)—This is a synthetic vasopressor which is chemically  $\beta$  hydroxy  $\beta$  (2,5 diethoxyphenyl) isopropyl amine. It can be autoclaved is non irritating and is a potent agent with a rather prolonged effect which comes on two minutes after intravenous injection and may last up to an hour.

It causes a decreased cardiac output, an increased peripheral resistance, and a rise in the right auricular and ventricular pressure and in peripheral venous mean pressure. Its action is peripheral on the arteries and veins. Pulse rate slowed an effect blocked by atropine. This may be a carotid and aortic sinus effect. Gives rise to no cardiac arrhythmia and seems to be safe in the presence of cyclopropane. Does not stimulate the higher centres. Pulmotor effect marked and causes desire to empty bladder.



**Methoxamine continued**

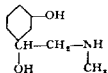
It reduces the urinary volume by depressing the glomerular filtration rate. Noradrenaline and phenylephrine do not share this effect. Must be used carefully in cases of hypertension, cardiac disease and hyperthyroidism.

Dosage must be kept small. 5-15 mg intramuscularly, 2 mg intravenously. Before repeat doses are given, important to see that effects of earlier injection have worn off. Delayed hypertensive effects have been observed. Put up in 1 ml ampoules containing 20 mg.

**Adrenaline**—Cannot be used as single injection to raise blood pressure as its effect is transient and also violent. Can be given as an intravenous drip as 1-250 000 solution in saline where 2 ml of 1-1000 adrenaline are added to 500 ml of saline (Frankis Evans). Blood pressure readings every five minutes determine the rate of drip (about 40-50 per minute) and must be continued in the ward after operation until the vasomotor system has regained its tone. This high blood adrenaline level may however interfere with the transmission of vasoconstrictor impulses from the vasomotor centre to the vessels because of ganglionic blocking. Adrenaline is an overall vasodilator and raises blood pressure by increasing cardiac output. In man it probably constricts the coronary arteries. Added to a solution of local analgesic in the theca it prolongs its analgesic action by 50 per cent. dosage recommended 0.25-0.5 ml 1-1000 solution (Braun 1900). No neurological effects have been blamed on this use of adrenaline. Adrenaline so injected is very slowly absorbed and does not contraindicate the use of cyclopropane to produce general anaesthesia. Noradrenaline 0.038 mg and neosynephrine 2 mg have also been successfully used to prolong spinal analgesia.

**Pholedrine** (Veritol Pholetone)—First used in 1937. It increases the rate and force of cardiac contraction but less so than ephedrine. Like ephedrine it delays destruction of adrenaline. It causes the spleen and liver to contract and increases the tone of the venous side of the circulation. Its action lasts nearly as long as that of ephedrine while repeated doses are almost as effective as the initial one. Side-effects are slight. Dose 4 to 5 mg intravenous, 20 mg intramuscular.

**Neosynephrine** (Phenylephrine Neophryn)—First described in 1931 by Kischinsky and Oberdisse. It causes no increase in heart rate and little improvement in the force of cardiac contraction. Chief



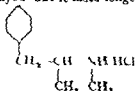
effect is to cause vasoconstriction by direct action on vessel walls and so can be used in place of adrenaline together with local analgesics for infiltration. Does not stimulate the cerebral cortex.

nor markedly relax the bronchi. Effect lasts 10-15 minutes but repeated injections have the same effect as the first one. Can be given on a continuous intravenous drip 1 ml (10 mg) in 500 ml of 5 per cent glucose the rate of drip varying from 200 to 50 drops a minute. Has no effect on conducting tissue of heart but may cause bradycardia from vagal stimulation so that this condition together with heart block are contra indications to its use. Does not constrict coronary vessels. There are no side-effects. Dosage  $\frac{1}{2}$ -1 ml of 1 per cent solution intravenously. Not contra indicated if cyclopropane is being used. If added to spinal analgesic solution (dose 2-3 mg) will prolong action of latter. Does not elevate blood sugar. It inhibits uterus and gut. It cannot be autoclaved.

**Phedracin** (Ciba 2020) — Introduced in 1938. Has little effect on heart. Produces vasoconstriction by direct action on vessels. Repeated injections not so effective as the initial dose. Causes erection of hairs and goose flesh with unpleasant tightness of the skin. Ampoules of 1 ml each contain 100 mg. Dose 100-200 mg.

**Noradrenaline** (Levophed Arterenol) — This can be given as an intravenous drip 2 ml of the 1-1000 solution being added to a pint bottle of 5 per cent glucose or normal saline plasma or blood. A continuous record of the blood pressure must be kept and the rate of flow adjusted after starting at 2 ml per minute. It must not be given along with deep cyclopropane anaesthesia may be safe with light cyclopropane. Its effects on cardiac output are not marked but it is a most powerful vasoconstrictor an action well marked on renal vessels. The rate of drip must be reduced gradually as it blocks vasoconstrictor impulses sent out from the medulla at the junction between the pre and post ganglionic fibres. Ephedrine may prevent this.

**Methedrine** (Methamphetamine Pervitin De oxyephedrine Desoxyn methyl amphetamine Norodin) — Allied to amphetamine (benzedrine) structurally is  $d\alpha$  methylamphetamine hydrochloride. Described in 1909 by Ogata. Increases cardiac rate and force of contraction and output. May cause bradycardia via vagal effect. Increases blood flow through kidneys. Produces vasoconstriction by direct action on vessels. Also stimulates respiration. Action long lasting and effective in repeated doses. Maximal effect 8½ minutes after intravenous injection. Compared with noradrenaline most of its effects delayed but it lasts longer. It elevates mood



reduces appetite and stimulates cortex. Has been used successfully to antidote acute barbiturate poisoning. It reduces post operative vomiting and is said to lessen nausea and vertigo after

*Methedrine continued*

fenestration operations Ampoules contain 20 mg per ml Dosage as prophylactic 15-20 mg intramuscularly If blood pressure falls unduly 5-10 mg intravenously with 15-20 mg intramuscularly Blood pressure should be checked before repeating dosage It may be dangerous in thyrotoxicosis and in heart disease

Methedrine ephedrine methoxamine and noradrenaline are the most effective drugs in this series

**Oenethyl** (2 methyl amino heptane) —A vasopressor drug allied to tuamine sulphate an aliphatic amine Produces rise in systolic and diastolic blood pressure due more to cardiac stimulation than to peripheral vasoconstriction If used during cyclopropane anaesthesia paroxysmal auricular tachycardia and ventricular extrasystoles may occur Dosage 50-100 mg—usually 75 mg

**Mephine** (Mephenteramine Wyamine) —This is N methylphenyl tertiary butylamine sulphate It is similar in effect to ephedrine but causes no ventricular arrhythmia and has no direct effect on the myocardium In dogs the coronary output is increased heart output not increased but blood pressure raised Does not increase heart rate and may slow it because of hypertensive effect on the aorticocarotid baroreceptors Does not stimulate the cortex No danger of cumulative effect Can be given intramuscularly when effect comes on in ten to fifteen minutes and lasts for one or two hours Effect after intravenous injection is immediate and lasts for thirty or forty five minutes Dose intravenous—5-15 mg slowly repeated when necessary 20-35 mg intramuscularly Has been stated to prevent adrenaline induced ventricular fibrillation during cyclopropane anaesthesia \*

Ether and cyclopropane if kept very light will raise the blood pressure slightly during spinal analgesia Oxygen is most helpful and may be given either alone or combined with 50-70 per cent nitrous oxide Carbon dioxide must not be given at all as there is usually too much of it in the body already in the hypotensive states when it might be needed Moreover the sympathetic block associated with spinal analgesia prevents impulses originating in the vasomotor centre owing to carbon dioxide being conveyed to the vessels while peripheral vasodilator effects of the gas predominate Trendelenburg's position is most helpful provided a hyperbaric solution has not just been injected Elevation of the legs empties their blood into the circulation—auto transfusion Finally intravenous fluid by helping the venous return to the heart is of paramount importance

Vasopressor drugs acting centrally are useless in high spinal analgesia as the effector pathways (the anterior roots of the spinal nerve) are out of action Such a drug is carbon dioxide

Gross hypertension may follow the use of ergot preparations given intravenously if the patient has already received a drug such as ephedrine as ergometrine (ergonovine) ergotamine and ergotoxin potentiate adrenaline and ephedrine

## PRELIMINARY MEDICATION

Inadequate premedication during spinal analgesia shows callous unconcern for the patient. It may also wreck the smoothness of an otherwise correct technical procedure.

The rapidly acting barbiturates pentobarbitone and quinalbarbitone in doses of 1½–3 gr given 1½–2 hours before operation are reliable and effective. Because of vasodilatation they may cause a slight fall in blood pressure. In addition morphine ½–1 gr with atropine ⅛–⅙ gr or with scopolamine ⅛ gr are usually given one hour before operation. The ampoule of omnopon ½ gr with scopolamine ⅛ gr (or half ampoule) is convenient for this purpose. In patients over 60 scopolamine may cause excitement and is better avoided. Patients who come to the theatre in an anxious state of mind should be helped by further intravenous doses of morphine.

Many anaesthetists prefer to give only light premedication such as morphine ½ gr with scopolamine ⅛ gr and to produce sleep during the operation by thiopentone in small doses supplemented by gas and oxygen or light cyclopropane if necessary.

The phenothiazine drugs give good pre-operative together with prolonged post operative sedation but cause in addition hypotension which may require a continuous noradrenaline drip for its control.

## ARMAMENTARIUM

The sterile trolley will vary according to individual requirements but will usually contain gloves gown and towels, lig iodine swabs and swabholder. A fine intradermal needle for raising the preliminary weal together with a sterile ampoule containing 1 per cent novocain and a small syringe. A large cutting needle mounted in a cork to puncture the skin. A spinal needle with short bevel to avoid partially puncturing the dura. The Howard Jones type size 21 s w g is a useful one. Finally ampoules of the drug to be injected syringe to inject it with ampoule of blood pressure raising drug. File to open ampoules.

Sterilization is most important and the whole outfit should be dry sterilized in a hot air oven or autoclaved. All glass syringes are ideal as they can be dry sterilized in a hot air oven without breaking readily. Sterile distilled water must be regarded with suspicion unless it comes from a fresh previously unopened container. Ampoules should not be stored in spirit or other antiseptic solution as minute faults in the glass may result in contamination of the contents and untoward results. The syringes and needles should be wrapped and put into a hot air sterilizer and maintained at 160° C for not less than an hour. If the autoclave is used the articles are kept at 260° F at a pressure of 27 lb/sq inch for thirty minutes. Boiling for five minutes in water cannot be relied on to kill the spores. Dry heat is not suitable for ordinary glass and metal syringes. Ampoules of lignocaine will stand several hours of autoclaving without change. Light and heavy nupercaine solutions show decreased potency if autoclaved for more than two hours while amethocaine is rather less stable.

### TECHNIQUE OF LUMBAR PUNCTURE

The lumbar puncture must be done in a good light on a table which can be tilted

**Puncture in Lateral Position**—The patient should be supported by a nurse and positioned as follows—

Back to be at edge of table and parallel to it

Knees to be flexed on to abdomen

Head to be brought down to knees

Hips and shoulders to be vertical to table to avoid rotation of vertebral column

Sudden movement to be avoided

In unilateral operations patient should be on diseased side when hyperbaric solutions on the sound side when hypobaric solutions are to be used. In the obese the median crease sags downwards sometimes as much as 1 in. so point of needle should be inserted above crease in these cases

The line joining the highest points of the iliac crests crosses either the spine of the fourth lumbar vertebra or the interspace between L 4 and L 5. Precise identification of the lumbar spines may be impossible but this does not matter so long as the first lumbar interspace (and those above this level) are avoided. When the chosen interspace is located the intradermal needle is inserted after careful palpation midway between the two spines and a small weal of novocain is raised. The hands of the anaesthetist have been scrubbed up and he has donned a sterile gown and gloves. The back has been painted over a large area with antiseptic and towels arranged suitably. While the skin weal is taking effect a syringe is filled with analgesic solution. A small incision is made in the skin with a large skin needle to prevent a tough skin from grasping the spinal needle tightly. Some prefer to use a Sise or a Rowbotham introducer as a cannula through which to introduce the spinal needle. The needle is then slowly pushed forward, parallel to the floor and at right angles to the back with its bevel in the plane to separate and not to divide the longitudinal fibres of the dura. If bone is met withdraw and slightly alter direction either upwards or downwards. The extradural space can be identified in many cases if a drop of analgesic solution is left on the hub of the needle as it is pushed inwards the negative pressure of the space causes the drop to be indrawn. From this point the dura is only a millimetre or two away. When the dura is pierced a click can often be felt. A successful puncture is followed by a free flow of cerebrospinal fluid on withdrawal of the stylet. Flow must be free not an occasional drop. Rotation of needle and pushing it in an extra millimetre will often ensure a free flow. Blood-stained cerebrospinal fluid is of no importance and usually becomes clear after a few ml have leaked away. Withdrawal of pure blood shows that needle point is probably in a vein and another puncture must be made. A dry tap is probably always due to failure to introduce the needle into the subarachnoid space.

Median approach easier than the lateral but if latter is preferred needle is inserted  $\frac{1}{2}$  in from the midline directly opposite the centre of the interspace and the needle is inserted at an angle of 25° to the midline. With this approach flexion of the back is not so important it is said to cause minimal pain and tough ligaments are avoided and so the sense of touch and needle control is more accurate. Sometimes it is successful when attempts using median approach have failed. In very fat patients bony landmarks may be impalpable and in such cases it is a good plan to raise three weals in the midline  $\frac{1}{2}$  in apart. If bone is struck when needle is inserted through one weal a successful puncture may be made if the others are used in turn. Another method to facilitate puncture in obese patients has been described \* the needle entering the relatively large space between the fifth lumbar vertebra and the sacrum. A weal is raised 1 cm medial and 2.5 cm superior to the medial superior aspect of the posterior superior iliac spine. This point is 1.5 cm lateral to the midpoint of the lumbosacral interspace and from it a needle is advanced 25° to the midline. This approach may also be used for lumbar extradural blocks. If the needle touches a root of the cauda equina the patient will complain of pain probably in the leg usually no harm results from this but if injection of the drug causes pain the position of the needle should be slightly altered. It shows that the needle point is within the vertebral canal and has pierced the ligamentum flavum. If failure results from puncture in one interspace it can often be made successfully if an adjacent interspace is used.

**Puncture in the Sitting Position**—Many workers find this easier than the lateral. Patient is placed across the table with his feet resting comfortably on a stool. Spine should be flexed with chin pressed on to sternum. Flexion of the spine rather than flexion of the hips is the aim. Puncture in this position is required for Kethenryton Wilson's technique with hypobaric solutions while it is convenient when block of the sacral roots by hyperbaric solutions is to be done although this latter block can be done equally well if the puncture is made with the patient in the lateral position provided that the caudal end of the patient is tipped downwards.

Puncture in the lumbar region requires no after treatment other than a dab with antiseptic to the skin. Infection does not occur in the skin and subcutaneous tissues.

## INJECTION OF THE ANALGESIC DRUG

The prepared solution is drawn up in correct amount into a suitably graduated syringe. It is beneficial to rinse out the syringe first with some of the solution which is later discarded. When crystals are used cerebrospinal fluid is allowed to drip into the ampoule and after solution has taken place the fluid is re-injected. During injection occasional aspiration of a small quantity of cerebrospinal fluid confirms that all of the solution reaches the subarachnoid space. The needle

### Injection of the Analgesic Drug *continued*

should remain in situ for a few seconds after injection to prevent leak of analgesic solution through the dural puncture hole

If the height of analgesia is to be controlled by the time a patient remains tilted levelling off should take place when sensory loss reaches two spinal segments below the desired level. This allows for a little spread with advancing time. Height of analgesia is tested with a needle or artery forcep. For almost any work inside the abdominal cavity except with the most gentle surgeons analgesia should reach to the subcostal arch (T 6-T 8) so that the table can be levelled when analgesia reaches the umbilicus (T 10). Upper abdominal procedures require block to T 2-5. The cough test is useful in estimating height of analgesia. The patient is asked to cough the relaxed part of the abdomen bulges out and any segment not relaxed remains firm and rigid.

### SERIAL OR CONTINUOUS SPINAL ANALGESIA

Introduced by Lemmon a Philadelphia surgeon in 1940. His theory was that after the nerve roots had soaked up their fill of analgesic drug much of the injected solution remained in the cerebrospinal fluid inactive from which it could be absorbed into the blood stream with the production of toxic symptoms. (It is now realized that absorption into the blood stream of small amounts of local analgesic solution is unlikely to cause toxic symptoms). Thus he devised a technique for making serial injections of minimal amounts of drug when required. After lumbar puncture in the lateral position with a nickel needle the patient is turned on to his back on to a special mattress overlying the operating table. In the mattress is a gap into which the hub of the needle projects. Novocain solution usually 5 per cent is contained in a syringe (5 ml) attached to a 30 in length of plastic tubing with a tap between syringe and tubing. All air is expelled and the free end of the tubing is connected to the lumbar puncture needle after removal of its stylet. After aspiration to test the fluid continuity of the system the initial dose of novocain is injected usually 100-150 mg. The tap is turned off to prevent leak of cerebrospinal fluid back into the tubing and syringe while the latter is fixed to the mattress at the head of the table with adhesive strapping. An average of 50 mg of novocain is required each half hour to maintain analgesia.

The method is very useful when either the scope or the duration of the operation is uncertain. It enables minimal dosage to be given without fear of inadequate analgesia and so is desirable in the aged the very young and the physically handicapped. Over 2000 consecutive cases have been reported by Lemmon with only one of them requiring supplementary anaesthesia. Tuohy has modified the technique (1942). He inserts a special lumbar puncture needle which is very slightly angulated at its inner end (Huber point). Through it he inserts a plastic catheter and directs it either caudad or cephalad by means of the direction of the angulation of the needle. When an inch or two of catheter are inside the theca the needle is withdrawn over it and a syringe is connected to the other end of the catheter by a fine needle. No

special mattress is required. The effects of the analgesic can be rapidly removed by saline irrigations of the subarachnoid space. Extra equipment is required: mattress, tubing, malleable nickel needle, but the method is a good one and avoids disappointment through using a dose too small and avoids disaster through using a dose too large.

### SPECIMEN TECHNIQUES

#### 1 Heavy Nupercaine.—

Before injection the nupercaine solution should be diluted with an equal or greater volume of cerebrospinal fluid in the syringe.

a LOW SPINAL.—A small volume of hyperbaric solution spread by gravity and inclination of spine.

Block of S 2-S 5 (piles, anal fissure, etc.)—Lumbar puncture in L 4-5 interspace, patient sitting or lying lateral with definite caudad tilt. Injection of 0.6 ml. slowly so that it trickles slowly into the bottom of the subarachnoid space. After one minute patient can lie supine as lumbar curve prevents spread upwards.

Block of S 1-S 5 (urethra, bladder neck, prostate, etc.)—Lumbar puncture L 3-4 interspace, patient sitting or lying on side with caudad tilt. Injection of 1 ml. slowly and patient lies level after one minute.

Block of L 1-S 5 i.e. of lumbar and sacral plexuses.—For unilateral analgesia, sound side upwards, puncture in L 3-4 interspace, patient in lateral position with spine level. Inject 1.4-1.6 ml. slowly and maintain lateral position for five to fifteen minutes. Unilateral analgesia gradually spreads to the other side unless patient is maintained in lateral position throughout operation. Suitable for operations on leg.

For bilateral blocks make injection with spine tilted 5° head down to prevent solution from accumulating in sacral curve. Immediately after injection turn patient on to back and level table.

b MID SPINAL.—For lower abdominal analgesia (T 7-8 to L 4). Puncture L 3-4 interspace, patient in lateral position with spine showing 5° head-down tilt. Inject 1.4 to 1.8 ml. solution and maintain position for five minutes for unilateral cases. For bilateral and intra-abdominal cases patient turned supine after injection and tilt maintained.

c HIGH SPINAL.—For upper abdominal analgesia (T 2-5 to L 4). Puncture L 2-3 interspace, patient in lateral position with 5° head-down tilt of spine. Inject 2 ml. solution after aspirating  $\frac{1}{2}$  ml. cerebrospinal fluid using a little barbotage. After injection patient turned supine and tilt maintained. No steep tilt allowed for 15 minutes after injection. Any excess solution pools at bottom of thoracic curve opposite roots of T 5.

#### 2 Hypobaric Nupercaine.—

a HOWARD JONES'S TECHNIQUE.—Depends on displacement of cerebrospinal fluid by a large volume of analgesic solution plus gravity to keep the solution in the desired place. Height of analgesia largely dependent on volume of solution injected.



Specimen Techniques—Hypobaric Nupercaine *continued*

Nupercaine and syringe should be warmed to body temperature. Patient lateral with affected side uppermost. Puncture L 2-3 or L 3-4. Inject solution slowly withdrawing a drop after each 3 ml to see that needle point is still in place. To calculate dosage measure spine from C 7 (vertebra prominens) to interiliac line. It varies between 15 in and 20 in. In males this distance in inches minus 4 gives number of ml to be injected to give analgesia to T 5. In females subtract 6 from the figure in inches. Thus 18 ml is maximum dose. A rough guide is to inject 10-12 ml for block to T 10, 12-14 ml for block to T 7-8, 15-18 ml for upper abdominal block T 2-5. For block of sacral nerves alone inject 6 ml at L 4-5 interspace. The needle is withdrawn after injection and patient is turned on to face to ensure soaking of posterior nerve roots. He is returned to supine position after six minutes with table in slight Trendelenburg position which is maintained throughout operation. If spine is kyphotic tilt must be more pronounced. For sacral block prone position is unnecessary.

Macintosh\* gives good reasons for assuming that the 1-1500 solution of nupercaine should be regarded as isobaric at room temperature and advises that it can be injected unheated with the patient in any posture after which he is positioned for operation. He believes it acts purely by volumetric displacement if this technique is followed.

A good result follows fairly rapid injection of the solution with immediate assumption of supine position with slight head down tilt. Good analgesia for upper abdominal operations is accompanied by inability to sit up or to cough effectively. There is loss of sensation as far as the nipple line or just above it.

- b **ETHERINGTON WILSON'S TECHNIQUE**—Depends on timing the ascent of the light solution up the vertebral canal with the patient sitting upright. A standard dose of 13-14 ml is injected at the L 4-5 interspace. A measurement between the spine of T 4 and the interiliac line is made with patient vertical; this gives the high spinal run. It is simpler to measure from C 7 to the interiliac line and then subtract 4 in. the average distance between the spines of C 7 and T 4. Having got the high spinal run in inches multiply it by the factor 5 and the resulting number is the time in seconds that the patient remains sitting after the start of the injection. This gives analgesia to T 5 or a little above. Injection must take 15 seconds; timing it by a stop watch and the patient is turned to the supine position smoothly but swiftly with an 8° head down tilt immediately at the conclusion of the calculated number of seconds. After lumbar puncture the flexed back is extended so that the patient sits bolt upright. The solution is warmed to body heat before injection. For mid spinals (L 10) patient remains sitting for three-quarters of the time for high block; while for low blocks the figure is

divided by one half. Thus if distance between C 7 and L 4-5 is 20 in. 20 minus 4 gives a high spinal run of 16. This multiplied by factor 5 gives a time of 80 sec. for a high spinal three-quarters of 80 i.e. 60 sec. for a mid spinal half of 80 i.e. 40 sec. for a low block. The author of this technique claims for it 85 per cent of perfect results.

Spinocain and duracaine 10 per cent novocain with alcohol to make it hypobaric is also used in this way. The dosage is  $1\frac{1}{2}$ -2 ml and the formula figure is 2 and not 5. With it analgesia is a little more certain but lasts a shorter time.

- c. LAKE TECHNIQUE \*—Patient lies prone on table and by pillows and tilting is arranged so that the T 7 spine is at the crest of the thoracic curve as seen against a black line on the theatre wall. Injection is made at L 2-3 interspace with patient prone. If the pressure of the cerebrospinal fluid is low compression of the internal jugular vein or straining will increase the flow. Previously warmed solution is very slowly injected at the rate of 3 ml. per minute and trickles up the posterior compartment of the dural sheath to its highest level. The patient remains in position for ten minutes and is then turned supine with a 5-10 Trendelenburg tilt. The volume of solution varies between 6 ml. for a small woman and 9 ml. for a large man. Duration of analgesia is  $1\frac{1}{2}$  hours ( $1\frac{1}{2}$  hours if 1-1000 solution is used). Extent of analgesia is from T 1 to L 3. The only afferent nerves remaining in the upper abdomen are the small branches of the phrenics which give rise to shoulder tip pain if the under surface of diaphragm is dealt with harshly together with afferent fibres of the vagus which give rise to nausea and faintness if stimulated by traction or by exaggerated peristalsis of the gut. Infiltration of the periosophageal tissue after opening the abdomen will block off the abdominal vagi.

UNILATLAL ANALGESIA WITH HYPOBARIC NUPERCALINE —Patient placed in lateral position side to be operated on uppermost. Warmed (105° F) nupercaine solution 12-14 ml injected in L 2-3 interspace. Lateral position maintained with slight head-down tilt. This method was described by Harris and Runk in 1937 and was recommended for operations on the kidney. Flaccidity of gluteal muscles of upper buttock indicate a successful result.

SEBRECHT'S TECHNIQUE —Devised by the great Bruges surgeon in 1934. Lumbar puncture performed in lateral position and then patient carefully moved to prone position with needle in situ. Hypobaric nupercaine 5 ml injected then pause for five minutes then height of analgesia tested. This procedure is then repeated until desired height of analgesia is reached. If there is marked bradycardia an interval longer than five minutes is allowed between injections. When correct level is reached needle is withdrawn and patient placed in position for operation. Slight Trendelenburg position throughout induction and operation.

Specimen Techniques *continued*

## 3 Procaine —

- a LUNDY'S TECHNIQUE** — No rule of thumb as to dosage but a rough guide is 1 mg per lb of body weight which takes into account both general and subarachnoid effects. A 10 per cent solution is used each ml containing 100 mg of novocain. Varying amounts of cerebrospinal fluid are used for dilution. Injections are given in the right lateral position but for obese and difficult patients sitting posture preferred. Height of analgesia is controlled by the amount of drug injected its concentration in the syringe and the interspace chosen for injection. Rate of injection is 1 ml per second.

For stomach and duodenum T 12–L 1 space 150 mg novocain with 3.5 ml cerebrospinal fluid

For abdominal gynaecology L 1–3 space 150 mg novocain with 3.5 ml cerebrospinal fluid

For inguinal and femoral hernia L 3–4 space 200 mg novocain with 1 ml cerebrospinal fluid

For suprapubic incisions L 3–4 space 120 mg novocain with 1.8 ml cerebrospinal fluid

For perineal operations L 3–4 space 80 mg novocain with 1.2 ml cerebrospinal fluid

For anal and rectal operations L 4–5 space 50 mg novocain with 1.5 ml cerebrospinal fluid

For transurethral operations cervix and penis L 4–5 space 80 mg novocain with 1.2 ml cerebrospinal fluid

- b MAXSON'S TECHNIQUE** — This employs gravity control. Puncture in L 4–5 interspace in sitting posture. Novocain crystals 200 mg for abdominal 100 mg for perineal operations. Rate of injection 5 seconds per ml. Amount of cerebrospinal fluid 4 ml for abdominal 2 ml for perineal work. For perineal operations patient kept sitting for 4 min after injection then supine on a flat table. For abdominal operations patient supine immediately after injection with table tilted 10° head down. When analgesia reaches costal margin table levelled. Head and neck raised on a pillow throughout.

- c LABAT'S TECHNIQUE** — Puncture in sitting posture from T 12–L 1 space downwards according to height of analgesia. After injection patient supine in Trendelenburg position. Neocaine crystals dissolved in 3 ml cerebrospinal fluid. Dosage 75–150 mg according to height of analgesia maximum 200 mg. Barbotage for upper abdominal cases of 8 ml (1 plus 1 minus 1 plus 1 minus 1 plus 1 minus 1 plus 1 minus 1 plus 1 minus 2). Less barbotage for lower levels.

Because of the short period of analgesia provided by procaine the drug is not used very frequently.

- 4 Amethocaine Hydrochloride** — This is used as crystals dissolved in cerebrospinal fluid as 1 per cent solution either alone or mixed with 10 per cent glucose as 1 per cent solution in 6 per cent glucose when it can be treated for dosage like hyperbaric

nupercaine (Silvertown's solution) When 1 per cent solution is mixed with one and a half parts of 10 per cent glucose sp g of mixture is 1013

- a **SISE'S TECHNIQUE**—Draw correct amount of 1 per cent solution into 5 ml syringe (1 ml = 10 mg) and add 3 ml 10 per cent glucose solution. Puncture in L 3-4 interspace and injection made in 30 seconds. Table put into 10° Trendelenburg and patient turned supine one minute from time injection was started. lessen tilt to 5°. In a further minute table levelled off and height of analgesia tested. Patient's head and neck raised on sandbag throughout proceedings. If on testing level is too high or too low alter tilt accordingly for a minute and re test before levelling off. Never leave patient in a tilted position for more than one minute without testing level of analgesia. For blocks of lumbar and sacral nerves alone after patient is turned on to back table is put into reverse Trendelenburg tilt of 3-4° and there maintained. Dosage is based on size and condition of patient. Small dose for frail old small patient. large dose for tough tall young patient. medium dose for average patient. For operations on anus amethocaine 8 7 6 mg (according to type)

For operations on perineum bladder legs vagina 14 12 10 mg

For hernia and appendix operations 16 14 12 mg

For lower abdominal operations 18 16 14 mg

For upper abdominal operations 20 17 14 mg

- b Amethocaine crystals dissolved in cerebrospinal fluid make a slightly hyperbaric solution. Dosage is one tenth that of novocain

- c **SPINAL D HEAVY**—Ampoules of 3 ml of 0.5 per cent amethocaine with 6 per cent glucose sp g 1035

For perineal block—1.5 ml injected with caudal end of theca below head end

For lower abdominal block—2 ml injected in lateral position with head end of spine tilted slightly downward (b). Patient placed in supine position with tilt maintained but with head raised on pillow

For upper abdominal block—2.5 ml injected in lateral position with head end of spine tilted slightly downward. Patient then placed supine with tilt maintained and head raised on pillow

- d **DINDALE'S TECHNIQUE**—Using isotonic Spinal D which is put up in 5 ml ampoules each containing amethocaine 0.4 per cent (20 mg) dextrose 4.6 per cent in water—sp g 1018 pH 5.0

For perineal block patient sitting and 2 ml injected. Position maintained five minutes

For lower limbs and groin patient in lateral position—flat and 3-4 ml injected

For lower abdomen 4 ml injected and patient put in 5° Trendelenburg position. methedrine intramuscularly 20 mg

For upper abdomen 5 ml injected patient put in 5° Trendelenburg position. methedrine intramuscularly 30 mg

This solution is hyperbaric but isotonic and so is unlikely to irritate the delicate nerve tissue

Specimen Techniques *continued*

- 5 **Metycaine**—Supplied in 2 ml ampoules of 10 per cent solution. Can be used like novocain but dosage to be a little less say three quarters of novocain dosage. Action lasts longer from 60 to 90 minutes.
- 6 **Lignocaine**—Solution of 5 per cent with dextrose 3.1 per cent has a pH of 6.5 and sp. g. of 1.018 and is a satisfactory agent for spinal analgesia with rapid onset and two hours duration. Should be diluted with cerebrospinal fluid before injection. Has been recommended for endoscopic prostatectomy given with the patient sitting in a dose of 1.5 ml when analgesia should extend to about T10\*. The 5 per cent solution is more potent than 0.5 per cent nupercaine solution and it tends to give a higher block because of diffusion within the subarachnoid space. It may result in a complete and unpleasant numbness with absolute loss of the sense of touch †.

**Differential Spinal Block.†**—When a dilute solution of procaine (0.2 per cent) is injected into the subarachnoid space the small fibres (C type fibres) are blocked first and other fibres are affected in sequence according to their size and myelination.

The following fibres are blocked —

- 1 The autonomic efferents (a) vasomotor (b) sudomotor (c) visceromotor—i.e. the thoracic, lumbar and sacral outflow
- 2 Sensation of pinprick
- 3 Stretch afferents

The following are not blocked —

- 1 Fibres conveying touch
- 2 Position
- 3 Vibration
- 4 Pain (other than pinprick)
- 5 Somatic motor

For induction 10–20 ml of 0.2 per cent procaine in normal saline is injected slowly. A continuous drip (1–1.5 ml per minute) will maintain block. Useful in certain diagnostic and therapeutic procedures.

**Total Spinal Analgesia**—This has been re-introduced by Griffiths and Gillies of Edinburgh‡ as a method of providing the surgeon with an almost bloodless operation field. The technique causes block of all the vasoconstrictors (T1–L2) together with analgesia and relaxation. Procaine 150–200 mg dissolved in 4 or 5 ml of cerebrospinal fluid with a steep head down tilt or heavy nupercaine are the drugs used and general anaesthesia is induced by thiopentone and maintained with nitrous oxide and plenty of oxygen allowing spontaneous respiration as a guide to the oxygenation of the medullary centres. It is stated that the generalized vasodilatation and full oxygenation prevent either tissue or parenchymatous organ damage including the kidneys and

Adams B. W. *Anaesthesia* 1956 11 97.  
 † Walker O. B. & J. *Anaesthesia* 1957 29 52.  
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heart Posture is employed (1) to ensure adequate blood supply to the brain with the head down tilted (2) to allow drainage of blood into dependent parts such as the lower limbs

Contra indications to total spinal analgesia might include —

- 1 Conditions interfering with coronary blood flow (a) Coronary disease (b) Aortitis (c) Aortic valvular disease (d) Certain cases of congenital heart disease
- 2 Severe anaemia
- 3 Conditions associated with low blood volume (a) Shock (b) Malnutrition (c) Toxic states
- 4 Conditions interfering with oxygenation (a) Respiratory obstruction (b) Severe emphysema
- 5 Conditions causing decrease in cerebral blood flow—arteriosclerosis

A pressor drug to raise the blood pressure should be given if there is —

- 1 Alteration in rate or rhythm of respiration
- 2 Increase in the arteriolar capillary refill time
- 3 Sudden fall in blood pressure after stabilization
- 4 Cyanosis in the presence of adequate respiration

**Intraspinal Segmental Analgesia** \*—A solution of 1-1000 amethocaine is injected through a fine catheter which is passed cephalad in the theca for 15-25 cm through a needle To prevent shearing off of the plastic catheter it must not be withdrawn unless the needle is simultaneously withdrawn with it Such a dilute solution approaches cerebrospinal fluid in tonicity, baricity and pH hence local neurological sequelæ are unlikely Persistence of motor power in legs may benefit circulation and result in less post operative thrombosis similarly bladder disturbances should be minimal

### CONDUCT OF THE ANALGESIA

The patient should usually be lightly asleep if not he should be made comfortable on the table Blood pressure apparatus to be fixed to arm The oscillometer is very useful as no stethoscope is needed with its tendency to slip out of place During the procedure the patient may require his lips moistened or his face fanned Nausea may sometimes be controlled by deep mouth breathing If analgesia ascends high up the body consciousness may be lost as afferent impulses reaching the cortex become fewer and fewer Apparatus for general anaesthesia and for oxygen therapy should be at hand as also should an intravenous set and suitable pressor drugs It is the duty of the anaesthetist to exercise constant vigilance—of the circulation and of the respiration

### EFFECTS OF SPINAL ANALGESIA BOTH INTRA AND EXTRADURAL

**Nervous System.**—

**ORDER OF BLOCKING NERVE FIBRES**—(1) Autonomic fibres  
(2) Imperature fibres—cold before warm (3) Pinprick fibres  
(4) Fibres conveying pain greater than pinprick (5) Touch fibres

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**Effects of Spinal Analgesia—Nervous System *continued***

- (6) Deep pressure fibres (7) Somatic motor fibres (8) Fibres conveying vibratory sense and proprioceptive impulses

During recovery return of sensibility is in the reverse order. Phantom limb pain can occur in patients who have suffered from such pains and may require morphine or even general anaesthesia for its relief. The pain finally eases off when numbness disappears. Spinal analgesia may also exacerbate pain in patients who have a severe pain in a limb e.g. sciatica (Gwendolen Harrison 1951 and Leatherdale\*)

**Cardiovascular System**—Fall in blood pressure is chief manifestation. It is roughly proportional to height of analgesia and is usually absent when sacral segments are alone involved. Blood volume and plasma volume not significantly changed in spinal analgesia. Blood pressure depends on tone of myocardium and arteries, capillary resistance and an ample supply of blood returning to the heart. Spinal analgesia may interfere with the first two by its paralysing action on the sympathetic fibres of the anterior roots while respiratory depression reduces the output of the thoracic pump in returning venous blood to the heart. Blood pressure fall is usually shown in the first twenty minutes after injection. Spinal analgesia by reducing the venous return to the heart results in a reduction of blood to the coronary vessels which if they are sclerosed may prove serious.

**THEORIES OF CAUSATION OF FALL IN BLOOD PRESSURE ~**

- a Diminished cardiac output consequent on reduction of venous return to heart due to muscular paralysis and lack of muscular propulsive force on veins and to quiet breathing due to intercostal paralysis and consequent lack of activity of thoracic pump
- b Dilatation of post arteriolar capillaries due to paralysis of vasoconstrictors. It is seen in entire vascular area somatic and visceral where anterior roots are paralysed together with their sympathetic vasoconstrictor fibres. Compensatory vasoconstriction takes place in areas not anaesthetized. In high spinal blocks majority of vasoconstrictor fibres—including those to arm (T 2-10)—are paralysed hence low blood pressure. As a concentration of solution (e.g. procaine 0.2 per cent) less than that required to cause muscular relaxation or analgesia will produce sympathetic block vasoconstrictor paralysis is often complete even if sensory block is only up as high say as T 4. The warm dry arm with dilated veins is often seen in cases of high spinal analgesia.
- c Splanchnic dilatation
- d Paralysis of sympathetic nerve supply to suprarenal glands (splanchnic nerves) with consequent hypo-adrenalinæmia
- e Absorption of drug into circulation. This is much more likely to be a cause of hypotension after extradural than after intradural analgesia because of the larger amount of analgesic drug injected.

*f* Ischaemia and hypoxia of vital centres

Spinal analgesia may not cause much fall in blood pressure in the absence of surgical stimuli or bodily movement. Blood pressure drop below 80 systolic and 60 diastolic should be taken notice of, while systolic blood pressure below 50 is unsatisfactory. Blood pressure and pulse rate usually fall together; a rising pulse rate with a falling blood pressure is a bad sign. There is no haemoconcentration as in true shock. A palpable superficial temporal artery is a reassuring sign, while a palpable carotid pulse accompanied by adequate tidal exchange indicates that the patient is not gravely collapsed.

**TREATMENT OF LOWERED BLOOD PRESSURE**—Injection of pressor drug, intravenous fluid, oxygen inhalations, Trendelenburg position, elevation of the legs. Some or all of these should be used prophylactically in high blocks and other cases judged to be doubtful risks. Inhalations of carbon dioxide must not be used. The central action of the gas cannot make itself felt because of paralysis of vasoconstrictor nerve fibres, while the peripheral effect on capillaries reduces the blood pressure still further. Blood pressure and pulse rates should be charted on record cards at frequent intervals during the operation. The vasoconstrictor reflex produced by haemorrhage is abolished by spinal block, in proportion to the height of the block, so that the patient is unable to protect himself against this stress.

**Respiratory System**—During spinal analgesia breathing always becomes slow and shallow, depending on the extent of motor block. Formerly this was thought to be accompanied by hypoxia and accumulation of carbon dioxide in the tissues. Recent work has not supported this. If there is any effect on the phrenic roots, patient cannot talk but can whisper. Such a condition requires oxygen immediately. Should respiratory paralysis occur, efficient artificial respiration must be started using the anaesthetic machine, a mechanical respirator or mouth to mouth breathing. This last method is done by holding an ordinary anaesthetic face piece over the patient's face, seeing that the airway is clear (by an endotracheal tube if necessary) and periodically blowing air into the patient's lungs from the anaesthetist's lungs. Expiration results from the normal elasticity of the chest. It will probably have to be continued for one or two hours depending on the drug injected.

**Gastro intestinal System**—The small gut is contracted as the sympathetic dilator impulses are removed, the vagus being all powerful. Sphincters are relaxed and peristalsis is active. Handling of the small bowel by the surgeon may cause it to dilate, as may the injection of atropine before operation. Nausea and vomiting due to the hypotension may occur and usually come on in waves lasting a minute or so and then passing away spontaneously. The spleen enlarges two or three times in high block when its sympathetic efferent fibres (splanchnic nerves) are paralysed. Stimuli arising in the upper abdomen may ascend along the unblocked vagi and perhaps the phrenics and cause

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**Treatment of Collapse during Spinal Analgesia (Intradural and Extradural)**—Turn patient on to back. The anaesthetist the assistant and the surgeon each has a task to perform. The anaesthetist must see that adequate amounts of oxygen get into the alveoli. The assistant after lowering the head of the table gets an intravenous drip going using noradrenaline saline (2 ml 1-1000 solution to 500 ml) or he injects  $7\frac{1}{2}$  to 15 mg of methedrine intravenously. Lowering head of table raises central blood pressure and increases venous return to heart while elevation of the legs increases the volume of circulating blood. The surgeon confirms the absence of the heart beat and if the abdomen is open he massages the heart through the left diaphragm using a strong compression and a quick release if no aortic pulsation or cardiac activity is palpable. Transthoracic cardiac massage is preferable and should not be delayed if after one minute the transabdominal massage fails to restore cardiac activity.

The causes of mortality in spinal analgesia are —

- a Lowering of blood pressure to point where coronary arteries are not adequately supplied
- b Hypoxia of vital centres
- c Progressive upward paralysis of respiratory mechanism. This should not occur as intermittent positive pressure respiration will carry the patient on until the paralysis wears off
- d Occasional toxic reaction to drugs injected

**Sequelae —**

- 1 **HEADACHE**—Only seen after intradural block never after extradural. This is the most frequent after effect occurring in 3-20 per cent of patients. Never present during the operation usually coming on in first three post operative days. Usually worse in sitting position and frequent after small operations such as piles. Often occipital and associated with pain and stiffness in neck may be vertical or frontal. Headaches are not rare after simple lumbar puncture. Sicard first suggested in 1902 that cause might be leakage of cerebrospinal fluid into extradural space. A patent needle track has been shown in dura at necropsy eleven days after puncture. Loss of up to 10 ml of fluid during lumbar puncture probably has no effect on subsequent headaches.

Luncture with an unflexed back may reduce the incidence of headaches as the dural hole is not stretched open.

**DIAGNOSIS**—A post-spinal headache is probably caused by the method of analgesia if —

- a It is different from any headache previously experienced by the patient
- b It is initiated or made worse by adoption of the sitting or erect posture
- c It has occipital and nuchal components
- d It is relieved by abdominal compression—which raises the venous pressure

**THEORIES OF CAUSATION —**

- a Low cerebrospinal fluid pressure. The theory which is most popular to-day is that the choroid plexus is unable to secrete

**Effects of Spinal Analgesia—Gastro intestinal System continued**

discomfort Para oesophageal infiltration of local analgesic solution will prevent this

**THEORIES OF CAUSATION OF NAUSEA AND VOMITING —**

- a* Hypotension
- b* Increased peristalsis
- c* Traction on nerve-endings and plexuses especially from vagus
- d* Presence of bile in stomach due to relaxation of pyloric and bile duct sphincters
- e* Morphine
- f* Psychic factors
- g* Hypoxia

**TREATMENT** consists in attending to the hypotension and hypoxia if present deep breathing through the mouth reassurance and attention to general comfort supplementary anaesthesia with thiopentone and nitrous oxide-oxygen or cyclopropane etc if the condition persists or if the surgeon's work is being interfered with

**Genito-urinary System**—Sphincters of bladder not relaxed so soiling of table by urine is not seen nor tone of ureters not greatly altered The penis is often engorged and flaccid due to paralysis of the nervi erigentes (S 2 and 3) this is a useful positive sign of successful block Spermatorrhœa is sometimes seen Block of the nerves from T 11 downwards results in painless labour

The tone of the uterus is not greatly altered after spinal analgesia in pregnancy so that this and extradural block are not contra indicated then

**Broken Needles**—If a needle breaks the proximal part and the stylet should if possible be left in place to serve as a guide to distal part If proximal part has already been removed another needle is thrust along the track of the first one for purposes of localization Removal should be attempted at once With patient prone a portable X ray may be helpful

**DIFFICULTIES AND COMPLICATIONS**

**The Spinal that does not Take**—Usual cause is failure to deposit all of the analgesic solution in the proper part of the subarachnoid space May be due to difficulty with lumbar puncture displacement of needle point after successful puncture by the syringe movement etc use of a long bevelled needle which allows part of the solution to be injected inside part outside the subarachnoid space faults in the use of gravity tilts dosage etc to control level of block faults in the solution alkalinization etc idiosyncrasy or the so-called rachis resistance This last is a definite entity seen when serial spinal analgesia is used with doses kept minimal Injection of a normal dose may give inadequate analgesia a partial failure in the one dose method if more solution is injected after aspiration of a little cerebrospinal fluid analgesia will be perfect Excessive alkalinity of the cerebrospinal fluid is said to cause precipitation of the analgesic drug with consequent failure

space intrathecal injection of 30 ml. warm saline or 1 per cent glucose intravenous injection of 20-50 ml distilled water Good results have followed the intravenous injection repeated in twelve to twenty four hours of vitamin B<sub>1</sub> (thiamine) 5-25 mg Nicotinic acid 100 mg in 500 ml of saline given intravenously thrice daily is also beneficial Posterior pituitary extract has also been recommended because of its antidiuretic power 2 mg linguets of desoxycorticone acetate or 5 mg intramuscularly eight hourly for 48 hours may be helpful and dihydroergotamine 1 ml intravenously or intramuscularly perhaps repeated may be beneficial Oxygen inhalations may do good while carbon dioxide increases the cerebral blood flow The application of a sphygmomanometer cuff around the head from forehead to occiput and inflating it has given good results This increases the venous pressure in the scalp which is transferred to the superior sagittal sinus by the parietal emissary veins this rise in venous pressure is said to hinder the absorption of cerebrospinal fluid and to raise its pressure so easing headache \* Macintosh recommends that for periods as long as surgically possible during the first few days after operation the patient should lie prone in the head-down position Deutsch† has had good results after the intravenous injection spread over three and a half hours and repeated if required of 5 per cent ethyl alcohol in 5 per cent glucose in water This is said to increase cerebral vasodilatation and stimulate the choroidal plexuses to secrete more cerebrospinal fluid If cerebrospinal fluid pressure is thought to be high lumbar puncture fluid depletion intravenous glucose 50 per cent in normal saline 50-100 ml magnesium sulphate 50 per cent enema given per rectum 6 oz caffeine sodium benzoate 7½ gr intravenous repeated Simple analgesics short wave diathermy etc may also be helpful

2. **BACKACHE**—Probably not much more common after spinal than after general anaesthetics The less traumatic the puncture the better A small pillow under the lumbar region reduces incidence of post-operative backache irrespective of method of anaesthesia Damage to intervertebral disk by the needle has been reported
3. **RETENTION OF URINE**—A little more common after spinal than after general anaesthesia Usually yields to carbuhol 4-1 ml intramuscularly repeated if necessary or neostigmine 0.5 mg intramuscularly Very occasionally prolonged retention due to spasm of vesical sphincter consequent on spinal analgesia is seen
4. **MENINGITIS**—Usually due to faulty asepsis but can occur with a seemingly flawless technique Distilled water gummed ampoule labels should be viewed with suspicion Autoclaving of the whole outfit is the ideal to be aimed at If all glass

Fry A. *Lancet* 1956 2, 890.

† Deutsch, Looch *Anesthesiology* 1952 12, Sept., 496.

Difficulties and Complications—Headache *continued*

sufficient fluid to maintain the cerebrospinal fluid pressure and this is made worse by all conditions producing fluid loss e.g. hemorrhage vomiting sweating lactation etc. The rate of leakage of cerebrospinal fluid exceeds its rate of formation and this results in changes in the hydrodynamics of the fluid with loss of cushioning of the brain and pressure or traction on vessels and sensitive brain structures basal dura tentorium etc. Pain arising from the tentorium cerebelli is transmitted by the fifth nerve that from structures on or below the inferior surface of the tentorium is transmitted by the ninth and tenth cranial and upper three cervical nerves. The negative pressure in the extradural space may draw cerebrospinal fluid from the subarachnoid space. In cases of traumatic leakage of cerebrospinal fluid the choroid plexus can form 500 ml per day. Patients who develop spinal headaches have a lower cerebrospinal fluid pressure than controls who have received a spinal but do not complain of headaches. Yet again cerebrospinal fluid pressures taken before spinal analgesia show that those patients who have a low value do not necessarily get headaches. Schaltenbrand considers that post spinal headache is due to reflex aliquorrhoea—reduction of CSF formation by the choroid plexuses resulting from physiological trespass.

- b High cerebrospinal fluid pressure—a response to meningeal irritation. The pressure of cerebrospinal fluid bears no constant relationship to systemic arterial blood pressure but varies directly with the intracranial venous pressure. Queckenstedt's test eases pain if applied to patients with low pressure headache makes it worse in those with high pressure headache. This is the mechanism of headache caused by chemical or bacterial invasion.

**TREATMENT**—This is prophylactic and combative. For the former the elimination of neurotic and unsuitable patients before operation including those with a history of frequent severe headaches. the use of a small needle (Anton 1923) or one with a conical tip separation rather than cutting on longitudinal fibres of dura by situation of bevel needle (Greene 1966) surgical and chemical cleanliness blocking foot of bed for 12–24 hours post operatively avoidance of strong light and also reading during this time. Low pressure headache is ameliorated by posture and by analgesics. Kaplan\* achieved good results by injecting 10–20 ml of normal saline into the extradural space immediately following the lumbar puncture. Treatment of established headache depends on cerebrospinal fluid pressure. if this is thought to be low the following measures may help frequent long drinks a tight abdominal binder injection of normal saline 25–50 ml into either the sacral or lumbar extradural

fully understood. It may well be the result of the drug injected while the low pH of a large volume of injected solution has also been blamed. Six grave cases of severe central nervous system sequelæ are reported by Bergner \* four of these ending fatally with incubation periods varying from 24 hours to 18 days. These analgesias appear to have been expertly managed with all right and proper precautions having been taken. Paralysis of the eighth nerve has been reported.

A constricting pachymeningitis may develop some time after subarachnoid block. Sequelæ may not be seen until many months after the performance of the block †

There is at present (1958) some emphasis on the serious neurological sequelæ of spinal analgesia and the pendulum of popularity is swinging away from the method.

### THE CHOICE OF SPINAL ANALGESIA

**Advantages**—Prevents the tough, strong patient from being soaked with muscle relaxant cheap, ideal for fit patients who object to being put to sleep lessens risk of vomiting causing pulmonary aspiration in patients with full stomach, quiet relaxed abdomen together with small contracted intestines and spontaneous breathing helps surgeon shock from surgical trauma lessened, upset of body chemistry minimal intestinal function returns early risk of explosion absent. Since the advent of muscle relaxants the need for spinal analgesia has decreased but it is still a useful method which has stood the test of time. Can be employed deliberately to produce hypotension and so less bleeding during operation.

**Disadvantages**—Operative mortality is slightly higher at least in high spinals than after general anaesthesia. It puts more of a strain on the cardiovascular system than a general anaesthetic by tending to cause hypotension. Some patients do not like the idea of it. The incidence of post operative headache. It does not reduce the incidence of post operative chest complications.

**Indications**—These vary greatly with different surgeons and anaesthetists. Specially indicated in strong muscular patients too tough for general anaesthesia. Useful in cases of acute bronchitis, active pulmonary tuberculosis, and bronchiectasis because of lack of the supposed pulmonary irritation still erroneously thought to be caused by even a skilfully given general inhalation anaesthetic. Kidney liver disease and diabetes because body chemistry is not interfered with. Acute cases with a full stomach may be less dangerous under spinal than general anaesthesia (stomachs cannot always be emptied by a stomach tube). Amputations through thigh or hip. Intestinal obstruction in the absence of acute distension or grave toxæmia. Paralytic ileus in the absence of acute peritonitis. Transurethral manipulations and in some

Bergner R. P. and others *Anesthesiology* 1951 12, No 6 Nov Dec.  
 † Rosenbaum, H. E. Long F. B. Hinchey T. R. and Trufant, S. A. *Amer med Ass Arch Neurol and Psychiatry* 1952 68 783.



Difficulties and Complications—Meningitis *continued*

syringes are used they can be placed with their needles in a hot air oven at 160 C for one hour before use. Infection with *B. pyocyaneus* has been reported and infection can occur with certain Gram negative bacilli difficult to cultivate on routine examination of the cerebrospinal fluid.

- 5 PARALYSIS OF SIXTH CRANIAL NERVE palsy of external rectus causing diplopia. Rare and usually disappears spontaneously in a few weeks. Onset usually between fifth and eleventh post operative days and associated with headache. It is only seen when the patient gets out of bed. May be delayed for three weeks while simple lumbar puncture without injection of analgesic solution can cause it. Has been said to occur in about 1 in 300 cases of spinal analgesia. Paralysis is never complete and is a different entity from the total paralysis associated with such conditions as skull fracture.

Other possible causes are (a) Mechanical due to upset of hydrodynamics of cerebrospinal fluid pressure causing stretching of the abducens nerve. Harvey Cushing pointed out that as the sixth nerve runs forwards from the posterior margin of the pons it is crossed by either the anterior inferior cerebellar or the internal auditory artery or by both so that if slight displacement of the cerebellum occurs these arteries are stretched and being fixed below to the basilar artery may cut into the nerve like a tight band. (b) Inflammatory low grade meningitis. (c) Toxic due to specific action of drug used acting on an unstable binocular vision mechanism. Phylogenetically a recently acquired one. A similar condition is seen in acute alcoholic intoxication.

When severe headache occurs steps must at once be taken to prevent diplopia. The patient must be sent back to bed and rehydrated both orally and parenterally. The antidiuretic hormone in posterior pituitary extract may be useful.

While the condition persists dark glasses should be worn with the outer one third of glass of affected eye made opaque. About 50 per cent of cases recover within a month. If after two years spontaneous recovery of function has not occurred operative cure may be considered. About 25 per cent of the cases show bilateral nerve involvement.

Paralysis of every cranial nerve except the first and tenth has been reported after spinal analgesia and transient deafness is not uncommon. Diplopia has been reported following general anaesthesia and after the use of relaxants and may then persist for some time.

- 6 OTHER NERVE LESIONS —Transient lesions of cauda equina causing abnormalities of leg reflexes incontinence of faeces retention of urine loss of sexual function sensory loss in lumbosacral distribution and temporary paralysis of peroneal nerve. Most of these clear up spontaneously. Radiculitis ascending myelitis adhesive arachnoiditis meningo-encephalitis and bulbar involvement have all been reported. Their cause is not

- f GENITO URINARY —Patients who may have an enlarged prostate (which is not the reason for the surgical procedure) Bladder difficulty may be complained of after operation  
If kidney function is poor the low blood pressure associated with spinal analgesia may result in temporary oliguria which may upset the subnormal renal function
- g CASES WITH DEFORMED BACKS —Because of difficulty in the performance of lumbar puncture
- h SKIN SEPSIS in lumbar region
- i NEUROLOGICAL OPERATIONS —In operations for lesions of the spinal cord or cauda equina on medico-legal grounds
- j IN CASES OF DEHYDRATION —These are bad risks and a much smaller dose of drug than usual is required

### SPINAL ANALGESIA IN NON SURGICAL CONDITIONS

**Therapeutic** —Patients with autonomic imbalance of the alimentary canal such as *megacolon* *cardiospasm*, etc. In the latter block must include the whole of the sympathetic outflow i.e. up to T 1. This is tested for by paralysis of the small muscles of the hands which occurs when T 1 is paralysed a patient cannot hold a piece of paper between his fingers. Relief of the condition by spinal block is an indication that sympathectomy may be helpful. The serial spinal technique of Lemmon is excellent for this high block. In megacolon block should reach to T 5. Patients with *eclampsia* are sometimes benefited by a high spinal block up to T 8 which reduces their blood pressure. Patients with *acute pulmonary oedema* due to left ventricular failure are said to have been successfully treated by spinal block which produces a bloodless phlebotomy caused by vasomotor paralysis. The vasodilatation results in a decreased venous return to the right heart and hence to the lungs relieving left ventricular failure. *Renal anuria* has been successfully treated by high spinal block which results in dilatation of renal vessels and increased secretion of urine. If the blood pressure falls too low phenylephrine or other blood pressure raising drug should be used. *Severe post partum hæmorrhage* has been relieved spinal analgesia increases uterine tone in labour.

*Severe cases of thyrotoxicosis* can be improved by high spinal analgesia which removes effect on thyroid of overactivity of supra renal glands by paralysing their splanchnic nerve supply.

*Reactionary hæmorrhage from prostate bed* has been stopped by spinal analgesia to S 1 which leaves intact the fibres producing vasoconstriction and contraction of the prostatic bed (L 1 and 2) blocking those fibres (S 2 3 4) causing dilatation of the vessels and prostatic capsule.

In cases of *embolism of the lower extremity* continuous spinal analgesia has been successfully employed a block to T 10 removing the vasoconstrictor fibres from the whole leg. By this means a block has been maintained for 50-60 hours. Continuous spinal analgesia lasting for fourteen days has been

Choice of Spinal Analgesia—Indications *continued*

cases of prostatectomy especially if done per urethram hemorrhoids when the surgeon requires an atonic sphincter. In operative obstetrics. For ligation of the inferior vena cava in cases of acute cor pulmonale. Five special indications are (1) When a bloodless field is especially desirable e.g. lumbar sympathectomy (2) When a contracted bowel (not seen with relaxants) is required e.g. abdomino perineal resection of rectum Hartmann's operation etc (3) In abdominal gynaecological operations in fat patients (4) For amputations in old arteriosclerotic or diabetic patients (5) In operative obstetrics because of its benign effect on the foetus and its protection of the mother from inhalation of gastric contents

✓ Contra indications—Should not without a good reason be pushed on to unwilling or unco-operative patients including young children. Often unwise in the following groups—

✓ a CARDIOVASCULAR—Severe shock hypotension with blood pressure below 100 systolic after injection of ephedrine hypertension when associated with myocardial weakness congestive heart failure patients unable to do reasonable physical work because of obesity senility myocardial degeneration toxæmia. Patients with a history of cardiac infarction should not receive a high spinal. If coronary disease is present a reasonable blood pressure is required to force blood into the coronary arteries. Such patients stand a low blood pressure poorly.

✓ Cerebral atheroma

b MECHANICAL—Patients with a splinted diaphragm which interferes with breathing such as hydramnios large ovarian and uterine tumours ascites omental obesity hypoxia is always a risk in these cases and oxygen should be given from the commencement of the analgesia if a spinal is used employing intermittent positive pressure respiration if necessary

✓ RESPIRATORY—Patients who are breathless from any cause these may become hypoxic especially if level of analgesia is high. On the other hand patients with emphysema or broncho spasm often do surprisingly well after spinal block.

✓ ABNORMALITIES OF THE CENTRAL NERVOUS SYSTEM—Spinal analgesia should not be given to a patient with an abnormality of the central nervous system whether it be congenital or acquired infective or degenerative active or inactive or healed. Any subsequent symptoms may be blamed on the spinal. Patients who are chronic sufferers from headaches will in all probability get a headache of moderate severity after operation. If it is suspected on the history (headache vomiting blurred vision) or the physical signs (papilloedema bradycardia drowsiness) that the patient has an expanding cerebral lesion a tumour cyst or abscess which may if the intracranial pressure is suddenly altered cause obstruction to the cerebrospinal fluid or blood-circulation (the pressure cone).

✓ GASTRO INTESTINAL PERFORATIONS—Contraction of the gut adds to the soiling of the peritoneum in these cases

Intrathecal ammonium sulphate 6 per cent in buffered solution with a pH of 7.2 is said to block the C type fibres carrying pain impulses from root irritation by metastatic growths in the cord. 3.5 ml of the 6 per cent solution should be mixed with cerebrospinal fluid and slowly re-injected.\*

Phenol in glycerin has been advocated instead of alcohol. It is hyperbaric. It should be proved by preliminary successful local analgesia with heavy nupercaine of the affected area that the needle is in the correct place†. Good results have followed the injection of 1 ml of 7.5 per cent phenol dissolved in myodil into the subarachnoid space‡.

### SPINAL ANALGESIA IN CHILDREN

Risk of circulatory depression minimal because of elasticity of their cardiovascular systems. Puncture should be in L3-4 interspace because cord extends lower in children than in adults. Dosage for analgesia to T8 is 10 mg of procaine for each year. Some prefer to use 1 mg procaine per lb of body weight. For a newborn baby 1-2 ml of 1-1500 nupercaine has been successfully used injected through an intra-venous needle. For pyloric stenosis in babies 20 mg of procaine in 1 ml cerebrospinal fluid. Specially indicated in shocking procedures such as open operations on hip joint. Useful for operations in the presence of acute respiratory infection, intestinal distension or a full stomach. Also in hot humid atmosphere to prevent ether convulsions. Lack of co-operation is chief drawback so premedication must be adequate. Small doses of morphine repeated as necessary make good sedative.

Etherington Wilson's dosage and timing by formula, using hypobaric nupercaine 1-2000 or 1-2500 —

Multiple	Distance of Intercostal Line to Spine of T4	Time in Secs	Dosage
5	3½ in	18	2 ml
5	4 in	20	2 ml
5	5 in	25	2½ ml
5	6 in	30	3 ml
5	7 in	35	4 ml
5	8 in	40	6 ml
5	9 in	45	7 ml
5	10 in	50	8½ ml
5	11 in	55	10 ml
5	12 in	60	11 ml

The above is for high spinal block to T5. For mid spinals to T9-10 time sitting up is three-quarters that for high spinal for low blocks up to L2 sitting time is one-half that for high blocks. Dosage does not vary in a given patient the injection takes 15 seconds and is included in the time that the patient is allowed to sit up.

See also articles by Slater§ and by Berkawitz||

Ludoji H, B. D. Lates, N. and Bishop L. *Anesthesiology* 1944 5 361

\* Mader R. M. *Lancet* 1955 1 28

† Ludoji H. *J. W. Soc. U. T. G.* Edin 1954 1 6

‡ Slater H. M. and Stephen, C. R. *Anesthesiology* 1950, 11 Nov 79

§ Berkawitz, S. and Greene, B. A. *Ibid.* 1951 12, May 376

### Spinal Analgesia in Non surgical Conditions—Therapeutic continued

reported and that without serious complications \* In such cases continuous extradural analgesia would be better If after many hours an analgesic drug loses its effect substitution of another drug should be tried For the treatment of paraplegic clonus in patients who have already lost sexual function and bladder control 5-10 ml of absolute alcohol should be injected after 5 ml of 5 per cent procaine solution This also helps to establish the automatic bladder †

### Diagnostic —

- 1 **INTSTINAL OBSTRUCTION**—Can be differentiated into organic and functional by spinal analgesia if functional contraction of the gut with relaxation of sphincters results in passage of gas and faeces within twenty minutes Differential spinal block using 0.2 per cent procaine solution can be used This concentration will give paralysis of the sympathetic fibres running to the splanchnics without any sensory (or motor) effect (Large bowel nerve supply *parasympathetic* vagi and S2-3-4—motor *sympathetic* T5-L3—inhibitory) Neither opiates nor atropine should be given to these patients as both drugs inhibit the gut
- 2 **THROMBOANGIITIS OBLITERANS**—If the vasoconstrictor fibres supplying the lower limbs which come from T10-L2 are blocked there is an increase in skin temperature of as much as 8°C in normal legs and in the vasospastic types of this disease in the thrombotic types this increase does not take place and further surgery is not likely to be beneficial

When sympathetic fibres alone need to be blocked a weak analgesic solution can be used e.g. 0.2 per cent procaine or 0.1 per cent amethocaine hydrochloride in distilled water which is hypobaric A suitable dose of the latter is 6-12 ml (6-12 mg) which usually gives a sympathetic block up to about T8

- 3 **SPINAL ANALGESIA IN INTRACTABLE PAIN**—Subarachnoid alcohol injection which was recommended by Dogliotti in 1931 and by G. Todd in 1937 is sometimes helpful in incurable cases of malignant disease with severe pain below the groin and iliac crests i.e. in the lumbar and sacral distribution The patient is placed in the semi prone position with diseased side uppermost and head tilted downwards Alcohol (absolute) which must be previously autoclaved is injected between T11 and T12 or T12 and L1 the dose being 0.5 ml The semi prone position ensures a greater effect on the posterior roots than on the anterior with this hypobaric solution The position must be maintained for one hour The beneficial effect may not be fully apparent for a week About 10 per cent of patients get rectal or bladder disability or weakness of the legs but this is an improvement on the results after chordotomy Headache may follow but subarachnoid adhesions are not produced The procedure may have to be repeated

## SUPPLEMENTARY ANÆSTHESIA (For Intradural and Extradural Analgesia)

No patient should receive a spinal injection without having an open vein e.g. a Mitchell or Gordh needle or a drip. This is to enable acute hypotension to be rapidly reversed.

This may be (1) Planned from the beginning and may be given either before or after the spinal injection. (2) Given during the course of the operation because of the partial failure wearing off or extension of scope of the operation because of the emotional discomfort and anxiety of the patient because of persistent vomiting or restlessness.

- 1 Intravenous inhalation or a combination of the two may be used.

In a similar manner light ether or light cyclopropane can be given before the spinal injection and some workers before embarking on a prolonged operation prefer their patients to be intubated. By use of these methods the patient need never know that he has had a spinal analgesic.

Alternatively the spinal is given in the usual way and allowed to become fixed. When it is ascertained that its height is adequate general anaesthesia is induced and carried to a light plane. For operations below the umbilicus thiopentone makes an excellent supplement but for higher blocks an inhalation agent is preferred by many. It has the advantage that oxygenation can be kept adequate.

- 2 If the patient requires a general anaesthetic during the course of the operation it is important to avoid a stage of delirium during the induction, and this is best done by a little intravenous thiopentone. Then gas and oxygen ether or cyclopropane can be added. The use of gas and oxygen alone may be undesirable if any hypoxia is allowed to exist.

The author prefers to use minimal thiopentone in 2½ per cent solution injected as necessary into a non-clotting needle to this is added nitrous oxide 6 l per minute and oxygen 2 l per minute. An endotracheal tube may be desirable (a) if the airway is difficult to maintain without it. (b) If the operation is likely to last a long time especially if it takes place in the upper abdomen. If the tube is used cyclopropane or gas-oxygen and pethidine or trileve are suitable supplements. Intravenous pentobarbitone (nembutal) 0.75 mg per ml is said to have the advantage over intravenous thiopentone that the patient is less confused on waking up.

Intravenous morphine ¼-½ gr may be sufficient to settle a nervous patient down if there is a little return of sensation towards the end of an operation. Intraperitoneal swabbing with 100 ml of ½ per cent novocain will also have a beneficial effect if mild pain stimuli are worrying the patient. During a hernia operation pulling on the sac may disturb the patient pain from this can often be abolished by injection of a little novocain into the neck of the sac. Similarly infiltration of the peri-oesophageal branches of the vagus at the cardiac end of the stomach

### SPINAL ANALGESIA IN OBSTETRICS

For Caesarean section method is unpopular as unexplained deaths have occurred \* especially after the use of novocain Reason for these deaths may be that pregnant uterus splints diaphragm so that hypoxia is the dangerous factor There is evidence too that smaller doses are required by women in labour than in normal patients Oxygen inhalations (100 per cent) from the beginning of the analgesia should avoid this danger Advantages are that baby is completely unaffected by the drug crying immediately after delivery while the uterine muscle contracts excellently owing to paralysis of sympathetic dilator fibres Premedication should consist only of atropine supplementary anaesthesia can be given after the delivery of the baby should it be necessary Analgesia should reach to the ninth thoracic dermatome The writer and his associates† have had considerable satisfaction from this method and recommend it They use 1.6 ml of hyperbaric nupercaine in the average case

Because of its lack of effect on the foetus and its power of producing absolute relaxation of the pelvic floor muscles spinal block to S<sub>1</sub> is excellent for forceps delivery 1 ml of heavy nupercaine being injected below L<sub>4</sub> with back level during and after injection In patients in labour the blood pressure must not be allowed to fall below 85 mm Hg

### SPINAL ANALGESIA IN UROLOGY

Very useful in cystoscopies and transurethral procedures If retrograde pyelography is to be done soon after cystoscopy block must not ascend higher than the roots of L<sub>2</sub> otherwise overfilling of the renal pelvis will not be prevented by the patient feeling pain For transurethral resection of the prostate 1.5 ml of lignocaine (5 per cent) given in the sitting position gives good results‡ In suprapubic prostatectomy some surgeons appreciate the relaxation and freedom from toxic effects obtained with spinal analgesia Others fear that reactionary haemorrhage may arise with the patient back in bed and his blood pressure returning to normal as the block passes off For nephrectomy or nephrolithotomy done with the patient in the lateral position hypobaric solutions should be used fat heavy patients kept in this position for any length of time tend to become hypoxic from interference with their respiration and thus often do better with general anaesthesia or spinal block accompanied by 100 per cent oxygen inhalations

### SPINAL ANALGESIA IN PREGNANCY

Spinal and extradural block in pregnancy do not materially increase the uterine tone and do not harm the foetus §

### SPINAL ANALGESIA IN THORACIC SURGERY

This is seldom if ever used to-day

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Williams B *Anaesthesia* 1958 13 448  
 † Thorn T C *Proc R Soc Med* 1954, 47 307  
 ‡ Adams B W *Anaesthesia* 1956 11 297  
 § Ruppert H L *Proc World Cong f Anaesth* 1956 16:1 Minneapolis B ges Publish  
 ing Co

the complete spinal nerves as they traverse the intervertebral foramina. Extradural block includes blocking of the sympathetic fibres travelling with the anterior or ventral roots which soon become the white rami. Usual distance between skin and extradural space 4-5 cm.

**Causes of Negative Pressure in Extradural Space**—There is a negative pressure in the extradural space. This is increased when the back is fully flexed but soon returns to normal (Janzen 1926, Heldt and Moloney 1928). Macintosh and Mushin have pointed out that the extradural space communicates with the paravertebral space which shares the general intrathoracic negative pressure during inspiration. Negative pressure in extradural space decreases from above downwards until in the sacral canal it is absent.

Another theory holds that the negative pressure is due to the dura being pushed anteriorly by the needle point, while yet a third theory holds that it is due to the flexion of the back causing lengthening of the space and so a negative pressure.

This negative extradural pressure may account for the leakage of cerebrospinal fluid into the extradural space after lumbar puncture.

**Technique**—When a solution of a local analgesic is injected into the extradural space it may exert its effect (1) On the nerve roots in the extradural space (2) On the nerve roots in the paravertebral spaces after they have shed their dural sheaths \* (3) On the nerve roots in the intradural or subarachnoid space after inward diffusion of the drug across the dura which diffusion has been proved to occur.

**FACTORS INFLUENCING SPREAD OF SOLUTION**—(1) The volume of solution injected (2) The age of the patient the old requiring less than the young (3) The force of injection—fast injection spreads the solution thinly over a wide area and may give an incomplete but extensive zone of analgesia (4) The drug used—lignocaine—appears to spread more widely than procaine etc (5) The level at which an injection is given (6) Gravity—a head-down tilt aids upward diffusion of the solution and vice versa. The height of analgesia produced by a given volume of solution is one of the great uncertainties of anaesthesia. Four segments on each side of the point of injection are said to be effected by the extradural injection of 10-15 ml of analgesic solution. The following solutions have been used—

#### **Local Analgesic Solutions Used.—**

Nupercaine 1-500 or 1-600 maximum dosage 60 ml. Analgesia is slow in onset—up to 20 minutes—but prolonged in effect—3 to 4 hours. Metycaine 1½ per cent solution.

Xylocaine 1 to 2 per cent which has a rapid onset in about ten minutes and gives good relaxation. Duration of effect 1½ to 2 hours—depending on strength of solution employed. 0.8 per cent solution gives good sensory without motor block. The



## Supplementary Anæsthesia continued

will do much to prevent sensations of nausea and faintness from worrying the patient in upper abdominal operations if he is conscious and to prevent hiccups if he is not

The use of phenothiazine derivatives such as chlorpromazine and promethazine 25 mg. of each with pethidine 50 mg. in the premedication will usually result in a calm and drowsy patient but fall in blood pressure extending into the post operative stage is a disadvantage and may be a danger

## EXTRADURAL BLOCK

**Definition**—Blockage of nerve-roots between the dura and the vertebral canal. A method giving reflex flaccidity of muscles analgesia and some intercostal activity together with a certain degree of hypotension and consequent ischæmia secondary to sympathetic blockade

**History**—Introduced by Corning and used in dogs by Cathelin and Sicard in 1901 and applied in clinical surgery by Pagès in 1921 and by Doghotti in 1931. Popularized in Britain by Massey Dawkins

**Anatomy**—The spinal dura mater represents the meningeal layer of the dura mater of the brain the periosteum lining the vertebral canal represents the outer layer of the cerebral dura. Between the spinal dura and the vertebral canal is the extradural (epidural peridural) space. Its average diameter is 0.5 cm. and it is widest in the *midline posteriorly in the lumbar region*

*Its boundaries* are superiorly the foramen magnum and inferiorly the sacro-coccygeal membrane posteriorly the anterior surfaces of the laminae and their connecting ligaments the roots of the vertebral spines and the ligamenta flava anteriorly the posterior longitudinal ligament covering the vertebral bodies and the disks laterally the pedicles and intervertebral foramina. The interspinous ligaments and the ligamenta flava dense gristly tissue are important in locating the extradural space

*The contents* include the dural sac the extradural plexus of veins and the spinal arteries lymphatics and fat. The veins become distended when the patient strains or coughs i.e. during bouts of increased intrathoracic pressure. There are fifty-eight intervertebral foramina and the degree of their patency is an important factor in controlling the height a given volume of analgesic solution will produce. They tend to be more permeable in the young than in the old so a volume of solution tends to give a higher block in the old than in the young

The dura mater is attached to the margins of the foramen magnum and so the extradural space has no communication with the cranium. It is also attached to the second and third cervical vertebrae and to the posterior longitudinal ligament. It ends at the lower border of the second sacral vertebra a point corresponding in level with the posterior superior iliac spines. Prolongations of the dura surround the spinal nerve roots and

Injection must only commence when position of needle point is certain

An initial test injection of 5 ml is made and if in five minutes there is no evidence of subarachnoid block such as inability to move feet the remainder of the solution is slowly injected frequent aspiration tests being made to avoid risk of subarachnoid or intravenous injection. Some workers are happy to persist in finding the extradural space at a higher or lower level and infiltrating it with solution even after the dura has been pierced.

The patient is then turned on the back with slight head down tilt for upper abdominal operations and slight foot down tilt for lower abdominal and pelvic proceedings. Dawkins recommends 1-600 nupercaine in half normal saline as a hypobaric solution if the head is to be tilted downwards. 1-600 nupercaine in 6 per cent glucose and normal saline if head is to be tilted up. This solution will gravitate to the caudal end of the theca should this space be entered by the needle.

A pressor drug may be given as there is likely to be some fall in the blood pressure but the ischaemia which results from this block may well be one of its most desirable features with which it might well be inadvisable to interfere. Sensory and sympathetic block are much more marked than motor block. Tactile sensation is not abolished unless xylocaine is used.

Onset of complete analgesia may require 10-20 minutes.

**Continuous Extradural Analgesia** \*—Greater control over duration and extent of analgesia can be gained if instead of a single injection of solution repeated injections are made through a plastic catheter introduced into the extradural space. The plastic catheter of 1 mm bore made of neoplex nylon or polyvinyl chloride is passed through a large needle the slightly angulated tip of which is accurately placed in the extradural space. The angle at the tip carries the catheter either up or down within the space according to the direction it is turned. This special needle designed by Tuohy though rather large in bore is relatively easy to insert.

**VOLUMES OF SOLUTION OF 1.5 PER CENT LIGNOCAINE REQUIRED**—For prostatectomy 10-20 ml for vaginal and perineal repair 20-40 ml for herniae appendicectomies etc 25-35 ml of solution are required for hysterectomies etc 25-40 ml for upper abdominal operations 35-50 ml For Caesarean section 15 ml taking care to control the blood pressure. In infants 5-6 ml of 1 per cent lignocaine has been reported to give good results †.

It is probable that a proportion of solution injected into the extradural space eventually diffuses through the dura into the subarachnoid space.

**Management of the Patient during Extradural Block.**—The general effects on the patient are similar to those described in the section on spinal analgesia.

**Extradural Block—Local Analgesic Solutions Used** *continued*

writer has had very considerable experience of the use of 1.5 per cent solution for surgical work and finds it most satisfactory.

Procaine 2 per cent with amethocaine 0.15 per cent maximum dosage 50 ml

Amethocaine hydrochloride can be added to lignocaine solution e.g. 50 mg added to 50 ml giving a 0.1 per cent strength. It increases the duration of analgesia by about 50 per cent.

Adrenaline is added in the usual strength i.e. 0.25 to 50 ml of solution.

**Method of Location of Extradural Space.**—The needle which should be of large bore (18–19 S.W.G.) or of smaller bore e.g. 20 S.W.G. according to preference rather blunt and short bevelled—to make recognition of differences of tissue easier—is inserted as for a subarachnoid block with the patient in the lateral position. For injection full flexion is bad as it stretches the dura and renders it more liable to puncture; it also reduces the extent of the extradural space. Full flexion should only be assumed when it is necessary to demonstrate the negative pressure. If the patient is very fat the sitting position is easier. The level at which the block is made is not very important. An easily palpable interspace should be selected; if possible a high one above L<sub>2</sub> for a high block and a lower space for a low block. Injection in the thoracic region is difficult and for it the patient should be sitting up and a negative pressure test employed rather than the loss of resistance test. The needle is halted after it has pierced the interspinous ligament. The following points suggest that the needle is in the extradural space:—

- 1 Sudden lack of resistance to advancing needle
- 2 Sudden ease of injection of a little local analgesic solution from a syringe attached to needle or injection of a little air. If point is in interspinous ligament plunger rebounds; if it is in the space plunger can be pushed in easily (Sicard and Forestier 1922; Dogliotti 1931). In the writer's opinion this is by far the best method.
- 3 Withdrawal of hanging drop of saline on hub of needle. Gutiérrez's sign (1932).
- 4 Movement of bubble on Odom's indicator (a glass tube with fine bore containing saline and an air bubble) which can be attached to hub of spinal needle.
- 5 Macintosh's extradural space indicator—a small rubber balloon attached to a record adaptor which is connected to the needle when it lies in the interspinous ligament. With a fine hypodermic needle air is injected into the thick rubber of the neck of the balloon and when the extradural space is entered the small balloon diminishes in size.
- 6 The Macintosh spring loaded needle.\*
- 7 The Iké syringe†. Both these last two automatically indicate by the release of a spring when the extradural space with its low resistance has been entered.

Macintosh, R. R. *Brit. med. J.* 1953, 1, 398.

† Iké, A. *Brit. J. Anaesth.* 1950, 22, 150.

artificial respiration with oxygen must be efficiently carried out with the use if necessary of a nor adrenaline drip (1-250 000) or other pressor drug

- 2 Time taken over the block
- 3 Time taken before onset of analgesia
- 4 Occasional backache caused by needle
- 5 Paralysis due to anterior spinal artery syndrome has been described \*

Has been used to control the pain of dissecting aneurysm and acute pancreatitis, to control eclampsia and to release vasomotor tone in the lower limbs e.g. after poliomyelitis †

This intriguing subject is more fully dealt with in *Spinal Epidural Analgesia* by Bromage P R Edinburgh and London E & S Livingstone 1954

## CHAPTER XVII

### REGIONAL ANÆSTHESIA

**History**—Modern local analgesia began with the introduction of cocaine into medical practice in 1884

Cocaine is the active principle of *Erythroxylon coca* a plant grown in South America isolated by Gaedlicke in 1855

Nieman a pupil of Wöhler purified and named the alkaloid in 1860

Von Anrep noted its local analgesic effect in 1878 Koller at the suggestion of Sigmund Freud proved its use in ophthalmology in 1884

Schleich (1892) and Reclus (1890) popularized infiltration analgesia while conduction analgesia or nerve block was discovered by Halsted and Hall in New York in 1884 The former as a result of acting as his own guinea pig during his researches on the new drug became a cocaine addict Arthur F Barker in 1899 was using infiltration analgesia at University College Hospital Braun introduced adrenaline about 1903

Substitutes for the toxic cocaine soon came Giesel's tropococaine appeared in 1891 Fourneau's stovaine in 1904 and Einhorn's novocain (procaine) in 1904

Meischer and Uhlmann introduced nupercaine in 1929

Lofgren and Lundqvist synthesized lignocaine in 1946 and Gordh used lignocaine in 1948

The hypodermic needle was described by Rynd of Dublin in 1845 by Pravaz Lyon in 1852 and by Alexander Wood of Edinburgh in 1855 The latter also devised a syringe and popularized hypodermic therapy

### GENERAL CONSIDERATIONS

Local analgesic drugs are mostly used as the soluble hydrochlorides It is probable that the alkalinity of the tissues frees the base and this unites with the nerve tissue Most local analgesics are esters of

Davies, A Solomon, B and Levene A. *Brit. med. J.*, 1958 2, 654.

† Allison, R. C. *Anæsthesia* 1958 13 157

**Management of the Patient during Extradural Block** *continued*

The blood pressure is likely to be higher in a conscious than in an unconscious patient. Most anaesthetists have their patients drowsy under the influence of an intravenous thiobarbiturate or under general anaesthesia e.g. gas and oxygen or cyclopropane. The blood pressure can be controlled by pressor drugs (*see p. 293*) the actual degree of hypotension depending on the amount of ischaemia thought desirable and the general condition of the patient. The popularizers of total spinal analgesia believe that given adequate ventilation and oxygenation a systolic blood pressure of 35 mm Hg is adequate as it is greater than the sum of the venous pressure and the osmotic pressure of the plasma always provided that peripheral resistance is abolished by the vasodilatation consequent on sympathetic block\*. The blood pressure of the present writer begins to rise if that of his patients descends very much below 60 mm Hg. During periods of hypotension a head-down tilt should if possible be adopted. The phenothiazine drugs will increase hypotension given before or during the operation.

Breathing during extradural block is generally quieter and easier than under general anaesthesia. This may be a reflex bronchodilatation initiated by baroreceptors stimulated by low blood pressure in the aortico-carotid sinuses or it may be due to relative ischaemia of the mucosae of the bronchi consequent on the low blood pressure in their supplying vessels the bronchial arteries. During high extradural block, the respiratory minute volume may be low but this does not appear to be harmful to the patient. Very occasionally a total extradural block is produced by a reasonable volume of drug. This results in apnoea and intermittent positive pressure respiration may be required for a period of up to two hours.

**ADVANTAGES** claimed for the method as against spinal analgesia are —

- 1 Less fall in blood pressure than in spinal analgesia. This is rather doubtful. In suitable cases the hypotension produced may be one of the great advantages of the block.
- 2 Less danger of meningitis.
- 3 Absence of post-operation headache and urinary retention.
- 4 Prolonged post-operation analgesia—up to 6 or 8 hours after nupercaine or amethocaine.

**ADVANTAGES** as compared with general anaesthesia —

- 1 Protection of the patient from the afferent impulses of the operation.
- 2 Maintenance of spontaneous respiration.
- 3 Provision by one injection of analgesia of relaxation ischaemia and contracted bowels.

**DISADVANTAGES** are —

- 1 Difficulty of being sure of position of needle point with risk of subarachnoid injection of a large volume of solution. If a massive spinal injection (subarachnoid) is given by mistake

2 **CARDIOVASCULAR SYSTEM** — Acute collapse — primary cardiac failure — Feeble pulse and cardiovascular collapse bradycardia pallor sweating This type of intoxication may be due to a rapid absorption of the drug so that the cardiovascular system is involved before the drug has time to reach the brain

3 **ALLERGIC PHENOMENA** — Rare may take form of broncho spasm urticaria or angioneurotic oedema

Toxicity may occur as a result of simple overdosage by inadvertent intravenous injection or because of susceptibility of the patient to normal dosage Injection with a moving needle together with frequent aspiration testing minimizes risk of intravenous injection

**Premedication** — Adequate premedication is essential for successful local analgesia in major surgery A barbiturate should be given to prevent possible toxic effects from the drug used The subject is set out in the chapter on SPINAL ANALGESIA While a patient under the influence of a barbiturate is partially protected from the toxic effects of local analgesic agents he is made more susceptible to these effects if he is given ether

The average patient does well if given pentobarbitone gr 3 (100 mg) two hours before operation and papaveretum  $\frac{1}{2}$  g and scopolamine gr  $\frac{1}{10}$  one hour before Ill and old patients require less

#### **Methods of Local Analgesia —**

- 1 Infiltration analgesia to abolish the pain due to surgical intervention and to ease pain associated with the injection of irritant drugs The direct injection of drugs into the incision and wound
- 2 Field block The injection of a local analgesic so as to create a zone of analgesia around the operative field
- 3 Nerve block The injection of a solution of local analgesic drug into the nerve or nerves supplying the area to be operated on
- 4 Refrigeration analgesia (see Chapter XIX)
- 5 Intravenous local analgesia
- 6 Intra arterial local analgesia
- 7 Topical or surface analgesia

**Syringes, etc** — Small volumes of solution can be injected with ordinary 10-ml and 20-ml syringes but larger volumes require some type of self filling syringe to expedite injection

If a three way tap is interposed between a 10-ml syringe and its needle with the third arm connected to a transfusion bottle by a length of rubber tubing a useful home-made apparatus is at hand the bottle acting as a reservoir for the local analgesic solution

Useful types of apparatus have been invented by Dunn Pitkin and Hamilton Bailey

### **PHARMACOLOGY OF DRUGS USED IN LOCAL ANALGESIA**

- 1 **Cocaine** (Benzoyl methylecgonine hydrochloride) — A derivative of the nitrogenous base ecgonine Cocaine is extracted from the leaves of *Erythroxylon coca* a tree indigenous to Bolivia and

**General Considerations continued**

aromatic acids and amino alcohols Cocaine and metycaine are benzoic acid esters procaine amethocaine and benzocaine are para aminobenzoic acid esters Cinchocaine and lignocaine are not esters

With the exception of cocaine (a vasoconstrictor) and lignocaine (no effect on vessels) local analgesic drugs are vasodilators Vasoconstricting agents such as adrenaline are usually added to local analgesic solutions to delay absorption and also to prolong their action Adrenaline keeps the solution in contact with nerve tissue for a prolonged time and also prevents the sudden flooding of the circulation with local analgesic drug

Detoxication occurs in the liver and speed of destruction is a measure of their toxicity

Liver disease may increase the toxicity of these drugs

In healthy adults 2 g of procaine or 0.5–1 g of xylocaine or 150 mg of either amethocaine or nupercaine in dilute solution can be injected The toxicity of a solution of local analgesic increases as the square of the strength of the solution (Macintosh)

**Theories of Impulse Conduction along Nerve fibres**—Eccles\* describes the ionic hypothesis of nerve conduction according to which conduction is associated with the entry of sodium ions into the nerve-fibres followed by the migration of potassium ions outwards During the interval between these events the interior of the fibres becomes positively charged relatively to the exterior—a state of reversal polarization During recovery the ions reverse the direction of their movement Local analgesic agents may prevent this ionic migration and so may prevent conduction of impulses Other theories of action of local analgesic drugs include the enzymatic theory the hormonal theory and the Meyer Overton lipid theory

**Signs of Toxicity**—Due to (1) Special sensitivity of the patient to the drug used (2) A high blood concentration of the drug from any cause These are more common after topical analgesia of the upper air passages than after local infiltration or nerve block This is because absorption from the lung is almost as rapid as from intravenous injection Treatment consists in (1) Artificial respiration with oxygen or air (2) Intravenous injection of a pressor drug e.g. methamphetamine 10 mg (3) Intravenous injection of just sufficient thiopentone to control convulsions if present (4) The ordinary treatment for cardiac arrest

Reactions to vasoconstrictor drugs may include pallor anxiety palpitations tachycardia hypertension and tachypnoea

**1. CENTRAL NERVOUS SYSTEM**—Convulsive respiratory failure Central stimulation followed by depression restlessness tremor convulsions respiratory failure Barbiturates are best antidotes both for prophylaxis and treatment (Tatum 1925) although perhaps their greatest use is in the actual control of established convulsions e.g. thiopentone

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Cocaine is detoxicated in the liver About 10 per cent is excreted by the kidneys unchanged

A safe dose of cocaine for surface analgesia is 3 mg per kilo of body weight (0 mg per stone) with a maximum of 200 mg (Macintosh)

**ACUTE INTOXICATION**—The patient becomes restless anxious and confused the pulse becomes rapid and the breathing irregular the pupils are dilated and there may be vomiting Convulsions and coma may precede death from respiratory failure In other cases acute cardiovascular collapse occurs it should be treated with intravenous injection of a pressor drug lowering of the head and oxygen inhalations Cardiac massage may be required The symptoms due to nervous intoxication should be treated with intravenous barbiturates and artificial respiration The mechanism of such catastrophes is not fully understood and is not necessarily due to over dosage

It is used by natives in Peru and Bolivia to increase their ability for hard work They chew coca leaves The euphoria produced is one cause of addiction

**2 Procaine (Novocain Planocaine Ethocaine Neocaine Allocaine Syncaine Kerocain Servocaine Surocaine)**—Para amino benzoyl-diethylaminoethanol hydrochloride Formed by the reaction between chloroethyl diethylamine and sodium para amino benzoate Soluble in water and alcohol Synthesized by Einhorn in 1904 it is about one quarter as toxic as cocaine It is useful for injection but must be used in 20 per cent solution before it has any surface effect

For infiltration the strength used is 0.25 per cent to 1 per cent for nerve block 1 per cent to 2 per cent Dry procaine from ampoules should be used to make up solutions before use Procaine 5.5 per cent in water is isotonic with pH of 6.4

Corlette\* states that to become isotonic a solution of 0.5 per cent procaine requires 0.8 per cent sodium chloride solution a 1 per cent solution requires 0.7 per cent sodium chloride solution while a 2 per cent procaine solution requires 0.55 per cent sodium chloride To get 0.8 per cent sodium chloride 12 ml of water are added to 100 ml of normal saline to get 0.7 per cent sodium chloride 23 ml of water are added to 100 ml of normal saline to get 0.55 per cent sodium chloride solution 63 ml of water are added to 100 ml of normal saline

Novutox contains 2 per cent procaine and is prepared by a cold sterilizing process It is made in various strengths with and without adrenaline proctocaine contains 1½ per cent procaine with 5 per cent benzyl alcohol in oil and its relatively prolonged action is probably due to its destructive action on nerve fibres due to the presence of benzyl alcohol

Analgesia lasts from 45 to 90 minutes

Pharmacology of Drugs used in Local Analgesia—Cocaine *continued*

Peru which the natives chew for their stimulant effect. Cocaine is soluble in water and alcohol and is now used solely for topical analgesia. It is an excellent surface analgesic and has a vasoconstrictor effect 4 per cent being a suitable strength. It is toxic when injected.

**CENTRAL NERVOUS SYSTEM**—This is stimulated from above downwards —

In the cortex excitement and restlessness are caused and mental powers increased.

In the medulla increase in blood pressure and respiratory rate and vomiting may occur. Later there may be depression with coma or convulsions and death from respiratory failure.

It blocks nerve conduction when applied to peripheral nerve trunks or nerve endings terminal nerve fibres being blocked at a concentration of 0.02 per cent.

The sympathetic nervous system is stimulated and cocaine potentiates the responses of organs supplied by sympathetic nerves to adrenaline.

Depression follows stimulation. It raises body temperature by increasing muscular activity causing vasoconstriction and so reducing heat radiation and by its effect on the heat regulating centre.

**RESPIRATORY SYSTEM**—In small doses cocaine and procaine stimulate the respiratory rate. Larger doses may cause respiratory arrest.

**CARDIOVASCULAR SYSTEM**—Sudden cardiac standstill has occurred. Cases of severe cardiovascular collapse are also seen. Small doses increase the pulse rate raise the blood pressure and potentiate the effects of adrenaline on capillaries (dilatation or constriction) probably like ephedrine by inhibiting amin oxidase the enzyme destroying adrenaline. Cocaine unlike procaine potentiates the effect of adrenaline on the automatic conductive tissues of the heart and favours the development of ventricular fibrillation.

**EYE**—Mydriasis perhaps due to sympathetic stimulation there is blanching of the conjunctiva and irritation of the corneal epithelium together with excellent analgesia. Used as 4 per cent solution for analgesia. The pupil still responds to light after it has become dilated by cocaine. Eserine counteracts the mydriatic effect of cocaine and atropine increases it. It usually reduces intra-ocular pressure but occasionally acute glaucoma results from its use.

On muscle it has a curare like effect even when the motor nerve has degenerated. Large doses have an effect on the heat regulating centre in the diencephalon and cause pyrexia.

**ABSORPTION**—From all mucous membranes including the urethra and bladder. There is some evidence that stronger solutions are absorbed less readily than weaker solutions owing to the increased vasoconstriction they produce. In nose and throat surgery used in 4 per cent to 20 per cent.

great but cardiovascular and central nervous symptoms of toxicity have been described. It does not dilate the pupil or make the cornea misty. It is not a vasodilator. It may have a cerebral effect causing drowsiness and amnesia. Pentobarbitone given well beforehand protects against its toxic symptoms. The maximum safe dose is probably about 0.5 gm. Amethocaine hydrochloride can be added to lignocaine to prolong its effect. It is metabolized in the liver. Has been given intravenously in 40 mg doses at five minute intervals to potentiate the analgesia of the thiopentone-gas-oxygen relaxant combination and also intramuscularly in 250 mg doses in 2 per cent solution. (See pp 251-253)

4. **Nupercaine**— $\alpha$ - $\eta$  butoxy cinchonic acid diethylamino ethylene amide. Synthesized by Miescher in 1929. This is a complex amine derived from quinoline. It forms neutral solutions in water and alcohol. Solutions can be repeatedly boiled without loss of analgesic potency.

It is precipitated from solution by alkali but the addition of a few drops of dilute hydrochloric or acetic acid reverses the change. It is incompatible also with hydrogen peroxide, potassium permanganate and silver and mercury salts.

It is more toxic than cocaine and procaine but also much more efficient as an analgesic. Thus its effective dose is less toxic than that of either of the two other agents while the duration of analgesia is much longer usually 2-3 hours.

The maximal dose depends on the concentration of the solution but should not exceed 2 mg/kg or 1 mg/kg in highly vascular areas. Elderly or ill and feeble patients should receive less than this. Injection of 6 ml of 2 per cent solution (accidentally) into tissues has caused death.

The usual strength used for infiltration is 1-1000 to 1-3000.

5. **Amethocaine Hydrochloride** (Pantocaine, Pontocaine, Decicain, Butethanol, Anethaine, Tetracaine, Pantokain, Dikain)—Para butyl aminobenzoyl-dimethyl aminoethanol hydrochloride. Synthesized by Eiskb in 1928. A butyl group ( $C_4H_9$ ) has been substituted for one of the hydrogen atoms of the para amino group while two methyl groups ( $CH_3$ ) replace two ethyl groups ( $C_2H_5$ ) of the procaine molecule. It is a base forming salts with acids and melts at 150°C. Solutions prepared under sterile conditions remain sterile. Non irritating to tissues and causes no pain on injection. It is a vasodilator and has a quinine like effect on the heart. It increases parasympathetic activity and inhibits the action of cholinesterase. Soluble in water and alcohol. Ten to twenty times as potent as procaine. Can be used for corneal analgesia in 0.5 per cent solution. The solution can be boiled without deterioration but is rendered inactive by alkalis. In 1 per cent solution it can be used for surface analgesia while for injection the usual strength is 1-2000 to 1-4000. It is hydrolysed by pseudocholinesterase. Hydroxymethocaine is less toxic as well as less active than amethocaine.

*Pharmacology of Drugs used in Local Analgesia—Procaine continued*

Toxic symptoms are referable either to the central nervous system with convulsions or to the cardiovascular system. Treatment as for cocaine intoxication.

**CHLOROPROCAINE**—This is a new compound having little toxic effect, a longer duration of action than procaine with more rapid onset together with a higher therapeutic index than any commonly used local analgesic drug.\*

It is hydrolysed four times as quickly as procaine and like it by pseudocholinesterase. When a patient is suspected of having a low pseudocholinesterase level e.g. in jaundice, anæmia, malnutrition, poisoning by war gas or weed killers etc. procaine, amethocaine and chlorprocaine should be used carefully. Sold as nesacaine.

**EFOCAINE**†—This solution produces analgesia for from six to fourteen days duration. It consists of procaine base 1 per cent, procaine hydrochloride 0.25 per cent, butyl para aminobenzoate 5 per cent, polyethylene glycol 300 2 per cent, propylene glycol 78 per cent, sod. metabisulphite 0.1 per cent, phenylmercuric borate (1-25 000) and water 20 per cent. It is a viscous saturated solution of the water insoluble anæsthetic base in a water miscible organic solvent. When it comes into contact with body fluids there is a deposition of the active ingredients to form a drug store house and thus it exerts a prolonged effect. The procaine and the butyl para aminobenzoate potentiate each other. Serious neurological complications have followed its use such as toxic neuritis and myelitis. Pooling of the drug in one spot should be avoided. It is designed for nerve block—not local infiltration—and is likely to be useful to control post-operative pain e.g. inferior hæmorrhoidal, intercostal, ilio-inguinal etc. Because of its viscosity a bayonet catch between syringe and needle is required. A preliminary procaine wheal relieves burning pain immediately resulting from its injection. Because of its toxic possibilities the drug should seldom be used.

- 3 **Lignocaine** (Lidocaine, diethyl amino 2,6 dimethylacetanilide, Xylocaine)—The most commonly used local analgesic agent in the U.K. to-day. A basic amide synthesized by Löfgren and Lundqvist in 1943 in Sweden. First used by Gordh in 1948. Very stable, not decomposed by boiling acids or alkalis. Its effects come on quicker and last longer than those of procaine. It seems to spread over a wider field than equal volumes of other analgesic drugs. Solutions of 0.25 per cent for infiltration with adrenaline 1-250 000, 2 to 4 per cent for topical analgesia in surgery of throat, larynx, pharynx etc. For nerve block 1.2 per cent to 1.5 per cent with adrenaline and for extradural block 1.2 per cent to 1.5 per cent or less with adrenaline—up to 50 ml. No more than 30 ml of the 2 per cent solution should be injected at one time. Toxicity not

Foldes, F. P., and McNall, P. G. *Anæsthesiology* 1952, 13, No. 3, May-June.

† Ansbro, F. P. and others *Ibid.* 1952, 13, No. 3, May-June.

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5. **Amethocaine Hydrochloride** (Pantocaine, Pontocaine, Decicain, Butethanol, Anethaine, Tetracaine, Pantokain, Dikain) — Para butyl aminobenzoyl-dimethyl aminoethanol hydrochloride. Synthesized by Lisich in 1928. A butyl group ( $C_4H_9$ ) has been substituted for one of the hydrogen atoms of the para amino group while two methyl groups ( $CH_3$ ) replace two ethyl groups ( $C_2H_5$ ) of the procaine molecule. It is a base forming salts with acids and melts at 150°C. Solutions prepared under sterile conditions remain sterile. Non irritating to tissues and causes no pain on injection. It is a vasodilator and has a quinine like effect on the heart. It increases parasympathetic activity and inhibits the action of cholinesterase. Soluble in water and alcohol. Ten to twenty times as potent as procaine. Can be used for corneal analgesia in 0.5 per cent solution. The solution can be boiled without deterioration but is rendered inactive by alkalis. In 1 per cent solution it can be used for surface analgesia while for injection the usual strength is 1-2000 to 1-4000. It is hydrolysed by pseudocholinesterase. Hydroxyamethocaine is less toxic as well as less active than amethocaine.

**Drugs used in Local Analgesia—Amethocaine Hydrochloride** *continued*

The maximum dose is 300 m<sup>m</sup> or 2 mg per lb of body weight but if half this is not exceeded toxic signs are very unlikely—apart from surface analgesia. Large doses are unwise and maximum for surface analgesia should be 8 ml of 0.5 per cent solution given in two or three divided doses with an interval of five minutes between each dose. Absorption from the alveoli—when analgesia for bronchoscopy is being induced—is almost as rapid as that following intravenous injection. Cocaine is probably safer for this purpose as its vasoconstrictor effect retards absorption somewhat.

Its effect lasts longer than procaine but not so long as that of nupercaine roughly 1½ to 3 hours. Onset of analgesia slow. It is inactivated by iodine and mercuric chloride. It is mildly antiseptic. Its combination with adrenaline greatly reduces its toxicity. Death occurs from respiratory failure, toxic symptoms being similar in appearance and treatment to those of cocaine.

- 6 Metycaine** (Nethesine Piperocaine)—Gamma (2-methyl piperidine) propyl benzoate hydrochloride. Synthesized by McElvain in 1927. Soluble in water and alcohol. This synthetic drug is rather more toxic and lasts longer than procaine. It has a good surface effect in 2–5 per cent solution. Used extensively for producing extradural sacral analgesia in 1½ per cent solution.

Procaine and amethocaine should not be used when the patient is receiving a sulphonamide drug or para-amino salicylic acid. Metycaine, lignocaine and nupercaine do not contain the para-amino benzoic acid group.

- 7 Butyn** (p-aminobenzoyl di-n-butyl propanol sulphate butacaine sulphate)—Synthesized in 1918 by Adams, Kamm and Volwiler. Used as topical analgesic in eye and throat and nose surgery e.g. bronchoscopy (2 per cent).

- 8 Benzocain** (Anæsthesin)—A simple alkyl ester. A powder used as a surface analgesic in painful ulcers and wounds and in tuberculous laryngitis. Orthocain (orthoform) is a similar product. Rappaport's solution for long acting analgesia is 2 per cent benzocain base in 40 per cent methane in water.

- 9 Carbocaine**—This is di-N-methyl pipercolic acid 2,6-dimethyl anilide. Was synthesized in 1957 by Egner and Ekenstam. The hydrochloride is resistant to acid and alkaline hydrolysis, will withstand autoclaving and is soluble in water. Slightly more toxic than procaine, less so than lignocaine. It lasts longer than and spreads as well as lignocaine.\*

In addition ethyl alcohol, benzyl alcohol, ammonium salts and phenol have been used for the prolonged relief of pain.

**Methods of Comparing Local Analgesics** †—

1. For topical analgesia the drug can be tested on the human tongue, the human laryngeal reflex, the mammalian cornea, the frog's skin. The rabbit's cornea is a popular method.

Ulfendahl H. D. *Acta Anæst Scand* 1956 1 8. Mumford, J. M. and Gray T. C. *Brit J Anæst* 1957 24 210.  
† Goddes, I. C. *Brit J Anæst* 1955 27 609.

- 2 Tests for conduction analgesia —sciatic nerve block in the frog guinea pig or rabbit lingual reflex in the dog change in the alpha wave of the action potential of isolated nerve fibres spinal analgesia—intensity and duration
- 3 Tests for infiltration analgesia —subcutaneous intradural injection in animals and man As will be seen there is no really reliable method

### DRUGS USED FOR VASOCONSTRICTION

Vasoconstrictor drugs have been used in local analgesia since Braun introduced adrenaline about 1903

**Adrenaline** —The tartrate which is synthetic is used for injection, the hydrochloride which is of animal origin for topical application For infiltration it is probably unnecessary to use a strength greater than 1:200,000 Adrenaline does not keep well in solution discoloration indicates decomposition it can be autoclaved once but not repeatedly The usual amount added is 7 drops in 100 ml or 2.5 minims to the ounce (3j) i.e. 1-200,000 or 2 ml to a pint (560 ml) of saline giving a 1-250,000 solution irrespective of the strength of the analgesic solution It is probably unwise to inject more than 0.5 ml at one time

Glass ampoules are preferable to rubber stoppered bottles

Adrenaline must be used in very low dilution if at all in cases of thyrotoxicosis and hypertension It may produce pallor tachycardia and syncope It should not be used if chloroform trilete or cyclopropane are to be given as general anaesthetics in addition lest the combination should cause ventricular fibrillation Vasoconstrictors help to produce a dry operative field

**Adrenaline Antagonists** —These may be necessary in case of over dosage with adrenaline —

- 1 The ergot alkaloids which have as a side effect a stimulating action on smooth muscle (hence the danger of using ergometrine and a pressor drug together)
- 2 The benzyl chlorethylamines e.g. dibenamine
- 3 Yohimbine
- 4 The imidazoles e.g. tolazoline (priscol) phentolamine which is both adrenolytic and sympatholytic dose 5-10 mg also known as regitine and rogatine
- 5 The benzodioxanes first synthesized in 1933 by Fourneau Adrenolytic action rapid and not long lasting Dose 15 mg intravenously

In addition the cholinergic substances mecholyl and carbachol are true physiological antagonists

**Cobefrin** (Corbasil) —This is 1-(3,4-dihydroxyphenyl) 2-amino propanol A synthetic substitute for adrenaline and is used like the latter in 0.5 per cent solution instead of 1:1000 solution It is said to be less likely to produce the minor collapse sometimes seen when adrenaline is used Its pressor action is between one sixth and one half that of adrenaline while it is more stimulating to the myocardium It cannot safely be combined with



**Drugs used for Vasoconstriction—Cobefrin continued**

cyclopropane With 0.5 per cent procaine cobefrin is used in a final strength of 1-80 000 with 1 per cent procaine 1-40 000 with 2 per cent procaine 1-20 000 Cannot be autoclaved

**Phenylephrine** (neosynephrine)—Used to cause vasoconstriction during local analgesia 0.25 to 0.5 ml of 1 per cent solution added to each 100 ml of local analgesic solution Causes no cerebral stimulation or tachycardia

Vasoconstrictors should be used very sparingly in (1) Hypertension (2) Thyrotoxicosis (3) In out patients (4) In old age

**INFILTRATION ANALGESIA**

A wheal is raised with a fine needle and through this wheal a larger needle is used to inject the main bulk of solution Procaine in 0.5 per cent solution is ideal for this technique For painless skin incisions infiltration should be intradermal as well as subcutaneous A slow gentle technique is important and the solution should be injected while the needle is moving

**TRANSVERSE SECTION ANÆSTHESIA**

A name given by Russian surgeons to a technique in which a transverse disk of tissue of a limb is infiltrated from skin to bone with a dilute solution of an analgesic drug such as 0.5 per cent procaine in large volume Nupercaine 1-5000 or 1-10 000 has also been used

**FIELD BLOCK OF SCALP AND CRANIUM**

**Anatomy**—The trigeminal nerve supplies the anterior two thirds the posterior divisions of the cervical nerves the posterior third (Fig 51)

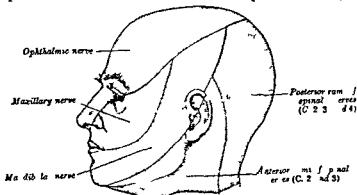


Fig 51—Showing the cutaneous nerve areas of the face and scalp  
(From *Gray's Anatomy* by kind permission of Professor T B Johnston)

There are four sensory nerves in front of the ear supratrochlear and supra-orbital both from the first division of the fifth nerve the zygomaticotemporal from the second division of the fifth nerve the auriculotemporal from the third division of the fifth nerve

The four sensory nerves behind the ear are the great auricular and the greater lesser and least occipital nerves from the cervical plexus

These nerves all converge towards the vertex of the scalp so that a band of infiltration passing just above the ear through the glabella and the occiput will block them all

**Technique**—Injections must be made in three layers—

- 1 The skin
- 2 The subcutaneous tissues
- 3 The periosteum

In addition solution should be injected into the substance of the temporalis muscle. The dura is insensitive except at the base of the skull

For removal of sebaceous cysts or suturing of small wounds the area is surrounded by a zone of infiltration. Periosteal injection is only necessary if bone is to be removed

### NERVE BLOCK FOR NOSE OPERATIONS\*

**Anatomy**—The nerve-supply is from the first or ophthalmic division and from the second or maxillary division of the trigeminal nerve  
In more detail—

The external nose is supplied by the frontal branch of the ophthalmic the anterior ethmoidal branch of the nasociliary (ophthalmic) the infra-orbital branch of the maxillary

The maxillary antrum of Highmore its lining is supplied by the maxillary nerve via the sphenopalatine ganglion

The frontal sinus frontal nerve branch of ophthalmic

The ethmoid region the anterior and posterior ethmoidal branches of the nasociliary

The sensory supply of the nasal cavities—fifth nerve—i.e. as follows—

The anterior one third of the septum and lateral walls by the anterior ethmoidal branch of the nasociliary nerve (division 1)

The posterior two-thirds of the septum and lateral walls by the posterior superior nasopalatine nerves from the sphenopalatine ganglion (division 2)

**Techniques.**—

- 1 **BLOCK OF MAXILLARY NERVE AND SPHENOPALATINE GANGLION**—Useful for operations on antrum (e.g. Caldwell Luc) and on upper lip palate and upper teeth as far as the incuspid

**ANATOMY**—The maxillary or second division of the fifth nerve is entirely sensory. It is given off from the middle of the Gasserian ganglion and passes forwards horizontally in the lower part of the lateral wall of the cavernous sinus until it leaves the skull through the foramen rotundum. It crosses the pterygomaxillary fissure to enter the orbit through the inferior orbital fissure and ends as the infra-orbital nerve

\*See also "Regional Anaesthesia for Surgery of the Nose and Sinuses" by Loftus Dale H.W. *Lancet* 1944 April 29 1 62 and *Local Anaesthesia Head and Neck* by Macintosh R.R. and Osler M. 1955 Edinburgh and London E & S Livingstone.

**Nerve block for Nose Operations—Techniques continued**

after emerging on to the face through the infra orbital foramen. The sphenopalatine ganglion of Meckel is situated in the pterygopalatine fossa in the upper part of the pterygomaxillary fissure lateral to the sphenopalatine foramen. Blocking of the nerve causes analgesia in the lateral nasal inferior palpebral and superior labial nerves the posterior middle and anterior superior alveolar nerves and the palatal nerves which supply the skin of the upper lip side of nose lower eyelid and malar region the teeth of the upper jaw and the underlying periosteum the mucosa of the maxillary antrum and of the hard and soft palate and the posterior part of the nasal cavity.

A wheal is raised 0.5 cm. below the midpoint of the zygoma which is over the anterior border of the coronoid process and through it a needle is introduced at right angles to the median plane of the head until it strikes the lateral plate of the pterygoid process at a depth of about 4 cm. Set marker 1 cm from skin surface and reinsert needle lightly anteriorly so that its point glances past the anterior margin of the external pterygoid plate and advances as far as the marker. The needle point should be in the pterygomaxillary fissure. The needle has been known to enter the pharynx or the orbit. If aspiration test is negative 3-4 ml. of 1.5 per cent solution of lignocaine is injected and a similar amount as the needle is slowly withdrawn.

Transient paralysis of the sixth cranial nerve may result it soon passes off.

- 2 **BLOCK OF ANTERIOR ETHMOIDAL NERVE (MEDIAN ORBITAL BLOCK)**—This is blocked in the medial wall of the orbit as the nerve passes through the anterior ethmoidal foramen. A wheal is raised 1 cm. above the caruncle at the inner canthus of the eye. A small needle is introduced along the upper medial angle of the orbit for 3.5 cm. keeping near the bone. Injection is made of 2 ml. of 1.5 per cent lignocaine.
- 3 **BLOCK OF FRONTAL NERVE**—From the same wheal as in anterior ethmoid block the needle is introduced more laterally towards the central part of the roof of the orbit where the frontal nerve lies between the periosteum and the levator palpebræ superior. Procaine 1 ml. of 2 per cent solution is injected in close contact with the bone.
- 4 **BLOCK OF INFRA ORBITAL NERVE**—The infra orbital nerve the terminal portion of the maxillary nerve divides at the infra-orbital foramen into inferior palpebral external nasal and superior labial branches. These supply the side of the nose the lower eyelid the upper lip and its mucosa. The infra orbital foramen is in line with the supra-orbital notch and canine fossa—both of which are palpable. It is 1 cm. below the margin of the orbit below the pupil when the eyes look forwards. The mental foramen is in the same straight line as is also the second bicuspid tooth. The foramen is 1 cm. below the margin of the orbit in line with the pupil when the eye looks forwards.

A needle is inserted through a wheal 1 cm below the middle of the lower orbital margin a finger breadth lateral to the ala of the nose. Procaine 2 ml of 2 per cent solution is deposited near the nerve as it issues from the foramen not while it is in the foramen. By this injection the upper lip and tip of nose are made insensitive.

For operations on the nasal septum bilateral maxillary and anterior ethmoidal block are necessary.

For radical operation on the antrum a maxillary block is indicated together with local infiltration inside the upper lip over the canine fossa.

For radical operation on the frontal sinus anterior ethmoidal and frontal blocks are necessary.

For operations for dacryocystitis anterior ethmoidal and infra orbital block are required.

### TOPICAL ANALGESIA OF THE NASAL CAVITIES\*

- 1 The nasal cavities are first sprayed with 10 per cent cocaine all excess solution being rejected and not swallowed. With a good light and a speculum the cavity is now packed with gauze soaked in equal volumes of 10 per cent cocaine and 1-1000 adrenaline. Cocaine is a powerful vasoconstrictor and so adrenaline although beloved of rhinologists is not really necessary. It causes moreover vasodilatation after the initial vasoconstriction has worn off. Trauma must be avoided. After ten minutes the packing is removed and the mucosa will be found to be avascular.
- A wool covered applicator is moistened with adrenaline and dipped into cocaine crystals and introduced so that it comes to lie against the sphenoid sinus between the posterior part of the middle turbinate and the septum. A second similar applicator is placed between the septum and the anterior end of the middle turbinate. After five minutes the patient is ready for operation.
- 2 Use of cocaine paste. Many formulae have been described a useful one is cocaine 7 gm thymol 60 mg dried suprarenal gland 15 gm liquid and soft paraffin equal parts to make 30 gm. For each patient 2 gm is used. The nasal cavities are first sprayed with cocaine solution (4 per cent) and then the mucosa of the septum and lateral walls is lightly painted with paste using a head mirror and light and a wool covered probe. Paste is applied to the area of the sphenopalatine ganglion behind the middle meatus and also to the area of the cribriform plate and acts on the long and short sphenopalatine nerves and the greater and lesser palatines in the first situation and on the anterior ethmoid nerve in the second.
- 3 MOFFETT'S METHOD †—The solution used is 2 ml of 8 per cent cocaine hydrochlor 2 ml of 1 per cent sod bicarb 1 ml of

See Macintosh, R. R. and Ostlere M. [*Local Analgesia Head and Neck* 1955 86 Edinburgh and London E & S Livingstone  
 † Moffett, A. J. *Anaesthesia* 1947 2 1

**Topical Analgesia of Nasal Cavities—Moffett's Method** *continued*

1-1000 adrenaline solution. A 2 ml syringe with bent needle is required

*Position 1* Patient lies on left side with pillow under left shoulder and head in lateral position at angle of 45° with vertical. One third of solution is drawn up, half being squirted into each naris along the floor of the nose.

*Position 2* After ten minutes second third of solution is drawn up and is similarly divided between the two sides of the nose. Patient pinches nose, turns prone, lies on face for ten minutes.

*Position 3* From prone position patient rolls on to right side as in Position 1 and remains ten minutes. If the septum is to be operated on 2 ml of 1 per cent procaine-adrenaline should be injected into the columella and base of septum in addition as this area is covered by squamous epithelium which will not absorb the topical agent.

The method gives good analgesia free from the unpleasantness of gauze packing and its resulting mild trauma. A modification of this technique has been well described by E. S. Curtiss\* who finds that excellent results are obtained by simply depositing 2 ml of the solution into the sphenoidal recess posterior to the middle turbinate bone on each side with the patient's neck extended until the head is upside down. Here the sphenopalatine ganglion and its branches lie and are bathed in solution lying in the superior meatus. The anterior ethmoidal nerve also becomes blocked at the same time. The procedure takes ten minutes. This has been again modified by Macintosh and Ostlere† who use Moffett's angulated cannula to deposit cocaine solution (2.5 ml of 5 per cent) on the inverted roof of the nose after preliminary spraying. Each naris is treated and the position is maintained for ten minutes. Excess solution must not be swallowed.

Nupercaine solution 0.5 per cent in 6 per cent glucose as used for spinal analgesia is a useful topical nasal analgesic.

**LINGUAL NERVE BLOCK** (J. Alan Carr)

The lingual nerve is the only sensory nerve supplying the floor of the mouth between the alveolar margin and the midline.

A finger in the retromolar fossa of the mandible will palpate the internal oblique line. The lingual nerve can be injected just medial to this line with 2 ml of 2 per cent procaine. A useful method of analgesia for removing calculi from Wharton's duct.

**VAGUS NERVE BLOCK**

This has been recommended by Mushin and others as a method of analgesia for broncho-oesophagoscopy.

\* Curtiss, E. S. *Lancet* 1952 **1** 989.  
† *Local Analgesia in Head and Neck* by Macintosh R. R. and Ostlere M. 1955. Edinburgh and London: E. & S. Livingstone.

**Anatomy**—Both motor and sensory roots spring from the medulla and leave the skull through the jugular foramen soon after its exit from the cranium it enlarges into a ganglion—the jugular ganglion—and after it is joined by twigs from the accessory nerve it again enlarges into the ganglion nodosum. It then passes down the neck in the carotid sheath lying between the internal carotid artery and the internal jugular vein. Then on the *right side* it passes between the first part of the subclavian artery and the right innominate vein runs along the side of the trachea to the back of the root of the right lung where it forms the posterior pulmonary plexus and is carried on to the oesophagus to form with the left vagus the oesophageal plexus. From this a single cord runs posterior to the oesophagus and enters the abdomen where it is distributed to the stomach coeliac plexus etc. On the *left side* the vagus enters the thorax between the left innominate vein anteriorly and between the left carotid and subclavian arteries. It crosses the left side of the aortic arch to the posterior aspect of the root of the left lung forming here the posterior pulmonary plexus. Branches leave this to form the oesophageal plexus after which the nerve continues into the abdomen.

The pharyngeal branch the chief motor nerve of the pharynx and the superior laryngeal branch arise from the ganglion nodosum. The recurrent laryngeal nerve arises on the left side just above the aortic arch and winds below the aorta. On the right side it is given off in front of the first part of the subclavian artery while the cardiac branches arise in the neck.

Vagal stimulation may cause arterial hypotension e.g. during pneumonectomy. The reflex may be abolished by curare.

The results of bilateral vagus block are (1) Tachycardia (2) Hypertension (3) Aphonia (4) Abolition of the cough reflex. In addition there may be signs and symptoms due to block of surrounding nerves e.g. (a) Horner's syndrome (cervical sympathetic) (b) Falling back of the tongue (hypoglossal) and (c) Dysphagia.

**Technique**—The needle is inserted through a wheal in front and slightly below the tip of the mastoid process so that its point lies on the anterior surface of the transverse process of the atlas. After aspiration 10 ml of solution (2 per cent procaine or 1.5 per cent xylocaine) are injected. As the hypoglossal nerve may also be blocked flaccidity of the tongue muscles may result and if bilateral may result in respiratory obstruction. A second method of vagus block at the jugular foramen is to raise a wheal anterior to the mastoid process just below the external auditory meatus. A 5-cm needle is then inserted perpendicularly to the skin until it touches the styloid process and then slips 2 cm behind it. Injection of a few ml of solution may also result in block of the glossopharyngeal accessory and hypoglossal nerves. This block should not be lightly undertaken but has been suggested for the treatment of cardiac arrhythmia and for pain in the larynx or pharynx. Bilateral block may be used for bronchoscopy and for operations on the larynx or pharynx.

**Vagus Nerve block continued**

**Superior Laryngeal Nerve block.**—This nerve is blocked at its point of division into the internal and external laryngeal nerve slightly below and anterior to the greater cornu of the hyoid bone

A wheal is raised over the thyroid notch in the midline the hyoid grasped between the thumb and index finger of the left hand and displaced laterally towards the side to be injected Through the wheal an 8-cm needle is introduced laterally and 2 per cent procaine solution is injected as the needle is advanced to the greater cornu—but not beyond it for fear of injuring the great vessels of the neck A further few ml of solution are injected as the needle is withdrawn A similar procedure is carried out on the other side through the same wheal

This block causes analgesia of the larynx above the cords so that food and drink must be prohibited for an adequate period depending on the drug and strength used

It is useful in conjunction with topical analgesia of the nose and pharynx to enable blind nasotracheal intubation to be performed for tracheo-bronchial toilet the coughing which results from irritation of the larynx below the cords soon passes off

**STELLATE GANGLION BLOCK\***

The stellate ganglion is formed by the fusion of the lowest of the three cervical ganglia with the first thoracic ganglion It is irregular in size and position being usually 1 to 3 cm long and differs in the same individual on the two sides Stellate ganglion block was first used for cerebral vascular accidents by Leriche and Fontaine in 1934 This should be named cervico-thoracic sympathetic block as when 10–15 ml of analgesic solution is injected into the correct plane at the base of the neck the middle cervical stellate and the second third and usually the fourth thoracic ganglia and their rami are blocked This results in interruption of all sympathetic fibres to most of the thorax head neck and arm (except the nerve of Kuntz) Certain visceral afferent fibres are also blocked e.g. the cervical cardiac nerves

**Anatomy**—It lies in front of the head of the first rib and the seventh cervical and first thoracic transverse process just behind the subclavian artery and origin of the vertebral artery It lies posterior to the carotid sheath on the longus colli and longus cervicis muscles It is anterior to the eighth cervical and first thoracic nerves so paræsthesia involving these nerves shows if stellate ganglion block is done from the front that the needle is too deeply placed On the right side of the apex of the lung and the dome of the pleura are anterior relations on the left side these structures are 1 in lower and so are not in such close relationship to the ganglion Vasoconstrictor fibres pass from the stellate and the other cervical sympathetic ganglia to a plexus around the internal carotid artery Twigs from the second and sometimes also from the third thoracic sympathetic ganglion often go directly to the upper extremity via the first thoracic nerve

thus by passing the stellate ganglion (the nerve of HUNTZ) But this nerve is usually blocked by spread of the analgesic solution down to the region of the fourth thoracic ganglion

It sends grey rami to the seventh and eighth cervical nerves gives origin to the inferior cervical cardiac nerve and supplies twigs to the vessels in its vicinity It may communicate with the vagus

#### **Physiological Effects of the Block.—**

- 1 Vasodilatation in head and neck vessels and in those of arm and hand
  - 2 Fall in intra-ocular pressure
  - 3 Inhibition of sweating salivary and mucous gland secretion in the bronchi
  - 4 Inhibition of cardiac pain and causalgic pain from upper extremity
- Its most frequent indication is to release vascular tone

**Indications**—Thrombosis embolism and spasm of vessels of the arm head (spasm after angiography) and neck e.g. Raynaud's disease Sudeck's atrophy hyperhidrosis causalgia of the upper limb pulmonary embolism status asthmaticus angina pectoris paroxysmal tachycardia head injury coma after intracranial operations Bell's palsy etc. auriculo-temporal syndrome tinnitus—some cases of eighth nerve deafness thrombosis of central retinal artery

**Technique**—Stellate ganglion block performed on a patient with an increased bleeding time or a decreased clotting time may result in a large hæmatoma in the deep planes of the neck Blocks for cerebrovascular accidents are done on the side opposite to that of the paralysed limbs Long acting drugs e.g. 6 per cent phenol or absolute alcohol are used chiefly to control cardiac pain Bilateral block should not be carried out at the same time

- 1 **PARATRACHEAL APPROACH (Moore\*)**—The patient lies supine chin forwards neck extended without a pillow Wheel raised two finger breadths lateral to the jugular notch and a similar distance above the clavicle which is on the medial border of the sternomastoid overlying the transverse process of the seventh cervical vertebra The position can be checked by palpating the tubercle of Chassaignac and the cricoid cartilage both of which are at the level of the sixth cervical transverse process i.e. a little higher than the wheel The needle 2 in or 3 in long is inserted directly backwards through the wheel while downward and backward pressure is exerted on the sternomastoid to draw the muscle and the carotid sheath laterally When contact is made with bone (C7) the needle is withdrawn 0.5 to 1 cm so that its point lies in front of the longus colli muscle and after careful aspiration for blood (the vertebral artery is very near) and for cerebrospinal fluid 10 ml of solution is injected This will if correctly placed diffuse up and down in the fascial plane and will block the ganglia and rami from C2 to T4 inclusive Thirty minutes may elapse before Horner's



Stellate Ganglion Block—Technique *continued*

syndrome and vasodilatation of the arm appear. This technique is in the present author's opinion the simplest and easiest.

- 2 ANTERIOR APPROACH (Walsh\*) —A number 1 needle is inserted medial to the sternal head of the sternomastoid just above and lateral to the sternal notch. The carotid artery is drawn laterally—the needle advanced at right angle to the skin lateral to the trachea on to the seventh cervical vertebra and its transverse process when 10 ml of solution is injected.
- 3 ANTERIOR APPROACH (V. Apgar†) —With the patient's head on a pillow and the face turned towards the sound side a needle is inserted through a wheal just above the clavicle and immediately lateral to the sternal head of the sternomastoid muscle. It is advanced directly backwards until it makes contact with the lateral part of the anterior aspect of the body of the seventh cervical vertebra. As the sympathetic chain lies on the longus colli muscle and not directly on the bone the needle is withdrawn 0.5 cm after which 5 ml of analgesic solution is injected. Horner's syndrome does not appear for ten to fifteen minutes. The carotid and jugular vessels are lateral to this point of injection.
- 4 ANTERIOR APPROACH (Alton Ochsner 1939) —With head rotated to sound side a wheal is raised 1 cm above and 1 cm medial to the midclavicular point. A needle is introduced medially at an angle of 45° to the midline avoiding a caudal inclination to miss the pleura. When the needle impinges on the body of the seventh cervical vertebra solution is deposited. The theca carotid and vertebral vessels must be avoided.
- 5 ANTERIOR APPROACH (Leriche Fontaine 1934) —Needle inserted through a wheal immediately above midpoint of clavicle and directed horizontally towards the transverse process of the first thoracic vertebra. As soon as bone is touched direction of needle altered 30° inwards and 20° downwards. Injection is made after the usual precautions of 10 ml of solution.
- 6 LATERAL APPROACH (Gonnard 1936) —From a wheal just above the midclavicular point a 10 cm needle is introduced medially at a sharp angle and contact is made with the upper surface of the first rib. The direction of the needle is then changed until its tip is guided along the rib surface to its medial extremity at its junction with the transverse process of the first thoracic vertebra. Here the injection is made after taking the usual most important precautions.
- 7 POSTERIOR APPROACH (Kappis Læwen Mandl 1947 Labat 1930 White 1940) —Patient suitably premedicated either sitting with head flexed or lying prone with head flexed. Spine of seventh cervical vertebra identified and wheal raised two finger breadths laterally at a point which corresponds with tip of transverse process of first thoracic vertebra.

Walsh, R. C. *B & med J* 1954 2 684.

† Apgar Virginia *Curr Res Anesth* 1948 27 49

Ten-cm needle introduced through wheel at right angle to skin until transverse process of first rib is touched. If distance is greater than 5 cm direction should be slightly changed. Tip of needle manipulated caudally until it slips off lower border of transverse process. Rubber marker set at 3 cm and needle inclined medially about 20° with median sagittal plane. A second bony contact should be made in 3 cm. If it is made at less depth needle should be partially withdrawn and introduced at a slightly smaller angle. If contact is not yet made at depth of 3 cm needle point must be directed slightly more towards the midline. Place a drop of solution on needle hub and ask patient to breathe deeply. A bubbling will indicate that needle tip is within pleura. When it is certain that injection will not be made into the pleura theca or a vessel 10 ml of solution is deposited in the close vicinity of the ganglion. This approach is the best one if a long acting drug is to be used and the position of the needle should be checked radiographically. Alcohol  $\pm$  5 ml or 6 per cent to 10 per cent phenol have given good results.

#### Signs of Successful Block.—

- 1 Horner's syndrome—miosis, enophthalmos and ptosis
- 2 Flushing of the cheek, face and neck and arm. Enlarged veins of arm
- 3 Flushing of the conjunctiva and sclera
- 4 Anhidrosis of the face and neck
- 5 Lacrimation
- 6 Stiffness of the nostril (Guttmann's sign)

#### Complications and Dangers of the Block.—

- 1 Pleural shock especially on the right side
- 2 Perforation of the oesophagus with infection
- 3 Intrathecal injection causing a total spinal block
- 4 Intravascular injection e.g. sending a volume of solution via the vertebral artery straight up to the medulla
- 5 Pneumothorax
- 6 Cardiac arrest—very rare
- 7 Alteration of voice from recurrent laryngeal nerve block
- 8 Phrenic nerve block
- 9 Brachial plexus block
- 10 Extradural block

Death has been reported after stellate ganglion block so that it should not be lightly undertaken. It is well worth doing however in cases of early cerebral thrombosis or embolism. It should be performed bilaterally and repeated several times.

### FIELD BLOCK FOR TONSILLECTOMY

**Anatomy**—The tonsil and its immediate surroundings are supplied by the middle and posterior palatine nerves and the glossopharyngeal nerve which gives off filaments which form a plexus called the *circulus tonsillaris*.

**Technique**—Half an hour before the analgesia is commenced an amethocaine lozenge (gr  $1\frac{1}{2}$ ) is given after which the mouth and pharynx should be sprayed with 4 to 10 per cent cocaine

**Field Block for Tonsillectomy—Technique continued**

solution some operators object to this preferring to keep the cough reflex active throughout

Injections of 3-5 ml of lignocaine and adrenaline are now made —

- 1 Into the upper part of the posterior pillar
- 2 Into the upper part of the anterior pillar Both pillars must be made cedematous throughout their whole extent
- 3 Into the triangular fold near the lower pole
- 4 Into the supratonsillar fossa after drawing the tonsil towards the middle line

The patient is sitting well supported in a chair Adequate time must be given for the analgesic to act Fainting is sometimes seen while the depression of the tongue by the spatula may cause discomfort

**Glossopharyngeal Nerve block** (Rovenstine and Papper) —Head fully rotated with patient lying supine At midpoint of a line joining the tip of the mastoid process to the angle of the jaw a needle inserted vertical to the skin makes contact with the styloid process 2 cm to 4 cm deep Needle partially withdrawn and re inserted 0.5 cm deep to and posterior to styloid process Injection of 6 ml of solution at this point will produce analgesia of posterior one third of tongue

Another technique for block of the glossopharyngeal nerve is to deposit solution near the jugular foramen A 5 cm needle is introduced through a wheal just below the external auditory meatus anterior to the mastoid process It is advanced perpendicularly to the skin until it meets the styloid process 1.5 to 2 cm deep and passes it posteriorly for a further 2 cm Analgesia involves the ninth to twelfth nerves inclusive and has been maintained with a long acting drug in cases of malignant disease in the posterior third of the tongue and in severe cases of neuralgia

**Suprascapular Nerve block \***—This arises from the fifth and sixth cervical nerves the upper trunk of the brachial plexus It runs laterally beneath the trapezius and omohyoid and enters the supraspinatus fossa through the suprascapular notch and below the superior transverse scapular ligament It proceeds laterally deep to the supraspinatus curves round the lateral border of the scapula to the infraspinatus fossa It supplies twigs to the shoulder joint the acromioclavicular joint and the supraspinatus and infraspinatus muscles The nerve is the sole pathway of somatic pain from the shoulder and acromioclavicular joints and structures surrounding them The block does not result in any skin analgesia

**TECHNIQUE**—Patient should be sitting with arms to the sides and head and shoulders slightly flexed With a skin pencil the spine of the scapula is lined in the inferior scapular angle is located and bisected by a line which crosses the first line A wheal is raised one finger breadth from the crossing in the

upper outer angle and a needle inserted downwards and medially to make contact with the bone of the supraspinatus fossa just lateral to the notch. Needle then withdrawn and re-introduced more medially until its point lies in the notch. Paræsthesiæ take the form of pain at the tip of the shoulder and after aspiration 10 ml of analgesic solution is injected. The block must be at the suprascapular notch as there the nerve is accessible to a needle and no afferent branches leave it before it passes through the notch. Types of shoulder pain relieved by this block include subacromial bursitis, painful abduction of the arm, calcified deposits about the capsule of the shoulder joint.

### CERVICAL PLEXUS BLOCK

This is paravertebral cervical analgesia.

**Anatomy**—Formed by the anterior primary divisions of the upper four cervical nerves, each one of which after leaving the intervertebral foramen passes behind the vertebral artery and comes to lie in the sulcus between the anterior and posterior tubercles of the transverse process of the appropriate cervical vertebra. Each nerve lies between the scalenus medius deeply and the levator anguli scapulæ under cover of the sternomastoid. Each of these four nerves except the first divides into upper and lower branches which form three loops lateral to the transverse processes. The loops are between C1 and C2, C2 and C3, C3 and C4. The lower branch of C4 joins C5 in the formation of the brachial plexus. The upper loop is directed forwards, the lower two backwards.

Branches are superficial (cutaneous), deep (muscular) and communicating.

**SUPERFICIAL BRANCHES** emerge posterior to the lateral border of the sternomastoid near its midpoint. They are—

1. **ASCENDING BRANCHES**—Small occipital (C2) and great auricular (C2 and 3). They supply skin of the occipito-mastoid region, auricle and parotid.
2. **TRANSVERSE BRANCH**—The nervus cutaneus colli (C2 and 3) supplying skin of anterior part of neck between the lower jaw and the sternum.
3. **DESCENDING BRANCHES**—The supra-acromial, supraclavicular and suprasternal nerves (C3 and 4) supplying skin of shoulder and upper pectoral region.

**DEEP BRANCHES** of the plexus are—

1. Phrenic nerve—C3, 4 and 5.
2. Anterior muscular branches.
3. Posterior muscular branches to sternomastoid, levator scapulæ, trapezius and scalenus medius.

**COMMUNICATING BRANCHES** are—

1. Sympathetic—each cervical nerve receives a grey ramus from the cervical sympathetic chain—the upper four nerves from the superior cervical ganglion.
2. Branch to hypoglossal nerve.

**Field Block for Tonsillectomy—Technique continued**

solution some operators object to this preferring to keep the cough reflex active throughout

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- 1 Into the upper part of the posterior pillar
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and above the upper border of the scapula posteriorly being supplied by the cervical plexus. Its chief indication is in thyroidectomy.

Cervical plexus block has been found useful in operations for the cure of oesophageal diverticula. Should debris be dislodged from the pouch as the cough reflex is retained it is unlikely to soil the trachea and bronchi. Bilateral phrenic block if it occurs is of no clinical significance.

Complications may include (1) Phrenic block (2) Intrathecal injection (3) Vagus and/or recurrent laryngeal nerve block causing aphonia (4) Cervical sympathetic block and Horner's syndrome.

### BRACHIAL PLEXUS BLOCK

**History**—Crile of Cleveland injected the plexus under direct vision in 1897.

Hirschel injected it blind through the axilla in 1911.

Kulenkampf after experimenting on himself devised the supraclavicular technique in 1912.

Patrick\* published his modification of the Kulenkampf technique in 1940. Macintosh and Mushin† describe this method in their excellently illustrated book. The method involves blocking the plexus as it lies on the first rib lateral to the subclavian artery.

**Anatomy**—The brachial plexus is formed from the anterior primary divisions of C5, C6, C7, C8 and T1.

It receives communicating twigs from C4 and T2. These nerves unite to form three trunks which lie in the neck above the clavicle. Each trunk divides behind the clavicle into anterior and posterior divisions which unite in the axilla to form cords.

The plexus is broad above and converges to the first rib.

Its anterior relations are the skin, superficial fascia, platysma and supraclavicular branches of the cervical plexus, the deep fascia and external jugular vein. The clavicle is in front of its lower part; the scalenus anterior is in front of its upper part.

Its posterior relations are the scalenus medius and the long thoracic nerve.

Its inferior relations are the first rib where the plexus lies between the subclavian artery anteriorly and the scalenus medius behind.

The plexus emerges from the intervertebral foramina and passes between the scalenus anterior and the scalenus medius. Close to their emergence the fifth and sixth nerves receive each a grey ramus from the middle cervical sympathetic ganglion. The seventh and eighth nerves each receives a grey ramus from the inferior cervical ganglion. The first thoracic nerve receives a grey ramus from and sends a white ramus to the first thoracic sympathetic ganglion. As the plexus converges on the first rib it is enclosed in a fibrous sheath contributed by the scalenus anterior and medius muscles.

**Cervical Plexus Block—Anatomy continued**

The posterior primary divisions of the cervical nerves supply skin and muscles of the back of the neck. Their cutaneous distribution spreads like a cape over the upper thorax and shoulders and this area is made insensitive in cervical plexus block.

Nerve supply of thyroid is from middle and inferior cervical sympathetic ganglion of the œsophagus the vagus and sympathetic of the trachea recurrent laryngeal and sympathetic of the sternomastoid the eleventh cranial and second and third cervical nerves.

**Technique**—The patient lies supine with shoulders slightly elevated and neck and head extended—as for thyroidectomy only with his head turned away from the side to be injected. Solution used is 1 per cent procaine or one of its congeners with adrenaline.

Deep cervical block requires the deposition of analgesic solution in the region of the transverse processes of the second third and fourth cervical vertebrae (the first sixth seventh and eighth nerves having no sensory branches).

Superficial cervical block is confined to the superficial branches of the plexus as they wind round the posterior border of the sternomastoid.

The following wheals are raised—

- 1 Just below the tip of the mastoid process
- 2 One finger breadth below wheal 1
- 3 One finger breadth below wheal 2. Each wheal is near the posterior border of the sternomastoid and corresponds with the transverse process of C 2 C 3 and C 4 respectively. The fairly easily palpable tubercle of Chassaignac—the anterior tubercle of transverse process of the sixth cervical vertebra—is a useful landmark.

Through each wheal a needle is inserted in a transverse direction posterior to the sternomastoid and seeks contact with a transverse process near its tip. This is not very deeply placed (about  $\frac{1}{2}$  to  $1\frac{1}{2}$  in). The needle must not be inserted deeply between or in front of the transverse processes for fear of piercing the dura or the carotid internal jugular or vertebral vessels. After a negative aspiration test for blood and cerebrospinal fluid and while the needle is in contact with the bone solution is injected as follows: 10 ml through the upper and lower needles and 5 ml through the middle one. The chief danger is intravascular injection.

Superficial cervical block is carried out by injecting 20 ml of analgesic solution between skin and muscle along the posterior border of the sternomastoid near its midpoint usually just below the position where it is crossed by the external jugular vein so as to cut off impulses from the ascending transverse and descending superficial branches of the plexus.

Cervical plexus block gives analgesia of the front and back of the neck the occipital region and a cape like area over the shoulders to below the clavicle the skin above the third

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\* *B. & J. Surg.* 1940 27 734.

† *Local Anæsthesia Brachial Plexus* 1944. Blackwell.



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and the lowest trunk of the brachial plexus. On the inner border between the grooves is the scalene tubercle. The subclavius muscle originates in front of the anterior groove and the scalenus medius is inserted behind the posterior groove. The lower surface has no costal groove, the inner border embraces the dome of the pleura while the outer border gives origin to the first slip of the serratus anterior.

**THE SUBCLAVIAN ARTERY** extends from its origin to the outer border of the first rib. The right subclavian comes from the innominate artery, the left from the aortic arch. At its highest point each artery is about 2 cm above the clavicle. Three parts of the artery are described: one medial, one behind and one lateral to the scalenus anterior.

The relations of the third part, i.e. the part lateral to the scalenus anterior, are: Anteriorly the skin, superficial fascia, platysma, deep fascia, descending branches of the cervical plexus, a plexus formed by the external and anterior jugular veins, and the transverse cervical and transverse scapular veins; the transverse cervical and transverse scapular arteries. Above and laterally is the plexus, while below is the first rib.

**THE SUBCLAVIAN VEIN** is separated from the plexus by the scalenus anterior. As it is well protected by the clavicle, it is unlikely to be punctured.

**Technique**—Patrick's aim was to infiltrate a sector of tissue lying between the mid-clavicular point on the skin and the first rib. He advocated starting the injections lateral to the plexus and slowly working medially until the subclavian artery pulsations transmitted along the needle indicated that the lower trunk of the plexus had been reached. Macintosh and Mushin modified this by starting the injections into the lower trunk close to the subclavian artery and working laterally. This modification is described below. The patient should be sitting or lying supine. The head is rotated to the other side and the arm and shoulder depressed. A wheal is raised 1 cm above the midpoint of the clavicle, a position—

- a Midway between the sternoclavicular and acromioclavicular joints
- b Crossed by a line produced downwards from the external jugular vein, made prominent by blowing out the cheeks
- c Just lateral to the pulsating subclavian artery, often palpable
- d Lateral to the outer border of the scalenus anterior, sometimes palpable under cover of the sternomastoid

A needle is inserted through the wheal downwards, inwards and backwards so that it is pointing to the spine of the second to fourth thoracic vertebra, a finger meanwhile guarding the subclavian artery and drawing it slightly medially. A cough from the patient is a warning that the pleura is being irritated by the needle. If paræsthesia is felt, the needle is steadied and 30 ml of solution is injected. The following nerves are blocked: The median, the musculocutaneous, the radial, the axillary, the ulnar, the medial brachial and antebrachial cutaneous.

**Brachial Plexus Block—Anatomy continued**

It lies first above and then to the outer side of the subclavian vessels and just above the clavicle lies between the skin and the first rib immediately behind the deep fascia

The upper trunk is formed by the anterior rami of C 5 and C 6

The middle trunk is formed by the anterior ramus of C 7

The lower trunk is formed by the anterior rami of C 8 and T 1

Behind the clavicle the trunks each divide into anterior and posterior divisions

The posterior cord is formed by the three posterior divisions

The medial cord is formed by the lowest anterior division

The lateral cord is formed by the upper two anterior divisions

Branches are given off from (1) Roots (2) Trunks (3) Cords

1 Branches from Roots —

Long thoracic nerve (of Bell) from C 5 6 and 7

Dorsalis scapulæ nerve from C 5

Muscular branches to the longus colli and the three scaleni

2 Branches from Trunks —

Suprascapular nerve (C 5 6)

Subclavius nerve (C 5 6)

3 Branches from Cords —

From lateral cord (three) Lateral anterior thoracic lateral head of the median musculocutaneous—all from C 5 6 7

From the posterior cord (four) Radial (C 5 6 7 8 and T 1) axillary (C 5 6) thoracodorsal (C 6 7 8) subscapular nerves (C 5 and 6)

From the medial cord (five) Medial head of the median medial anterior thoracic ulnar medial antebrachial cutaneous (all from C 8 T 1) medial brachial cutaneous (T 1)

**THE SCALENUS ANTERIOR** arises from the anterior tubercles of the transverse processes of the third fourth fifth and sixth cervical vertebrae. It is inserted into the scalene tubercle on the inner border of the first rib. The muscle lies anterior to the plexus being separated from it below by the subclavian artery. Its lateral border if it is palpable is a guide to the position of the plexus.

**THE SCALENUS MEDIUS** arises from the posterior tubercles of the six lowest cervical vertebrae and is inserted into the upper surface of the first rib behind the groove made by the plexus and the subclavian artery. The plexus thus lies in front of the muscle.

**THE FIRST RIB** lies in an almost horizontal plane being inclined slightly downwards and forwards. It passes below the clavicle at about the junction of its inner and middle thirds. Its surfaces look upwards and downwards and its borders inwards and outwards.

The head has a single articular facet which articulates with the body of the first thoracic vertebra. The tubercle articulates with the transverse process of the same vertebra.

The upper surface has two transverse grooves an anterior for the subclavian vein and a posterior for the subclavian artery.

components of the plexus. Analgesic solution injected into the tissue surrounding the plexus can thus ascend to block the phrenic nerve. Such a block is harmless and causes no symptoms even if bilateral but if the patient has a respiratory difficulty e.g. emphysema or kyphosis or if a general anæsthetic is to be administered the possibility of diaphragmatic paralysis must be borne in mind.

- 2 Puncture of vessels including subclavian artery. Hematomata may form but cause no trouble. Intravascular injection must be avoided.
  - 3 Pneumothorax. Trouble seldom occurs. It causes pain in the chest which may not come on for several hours. Surgical emphysema may be seen probably due to wounding of the lung by the needle. If a radiograph shows a large area of lung collapse air should be withdrawn from the chest. Bilateral pneumothorax may be a severe condition.
- Pain in the chest during needling may also be due to irritation of the long thoracic nerve. Silent pneumothorax is unlikely as air in the chest is usually accompanied by pain.
- 4 Toxic effect of drug injected. Slow injection lessens chance of this.
  - 5 Post-operative disability following brachial plexus block is most rare.

**LOOKMAN'S METHOD**—Lookman\* describes a closed fascial space which is an airtight and watertight compartment pyramidal in shape with apex pointing upwards and medially towards the exits from the spine of the fourth cervical nerve the base is the upper surface of the first rib between the attachments of the scalenus anterior and the scalenus medius muscles medial border formed by the fusion of the epineurium of all five roots of the brachial plexus with the epimysium of the scalene muscles and by dense connective tissue round the subclavian artery which is attached to the inner border of the first rib. The anterior and posterior borders are formed by the lateral borders of the scalenus anterior and medius muscles. The prevertebral fascia forms the lateral surface of the space. The anterior and posterior surfaces are formed by the opposing surfaces of the two scalene muscles between which the plexus and the artery lie. The contents of the space are the second and third parts of the subclavian artery together with the roots and trunks of the brachial plexus. The veins, dome of pleura and the stellate ganglion are outside the space. If a needle point lies behind the artery and strikes the first rib it will lie in close proximity to the plexus in this enclosed space.

**TECHNIQUE**—A number 1 needle is inserted as in the classical manner from a point 1 cm. above the midpoint of the clavicle lateral to the pulsating subclavian artery. The index finger guards and retracts the artery and the needle goes in just above the retracting finger in a backward downward and inward direction until it meets the upper surface of the first rib with its point between the artery and the plexus—30 ml

*Brachial Plexus Block—Technique continued*

If paræsthesias are not felt in the arm and hand after one or two needle thrusts the upper surface of the first rib is contacted and the needle inserted on to it so that the pulsations of the subclavian artery are transmitted to the needle. This is at a depth of  $\frac{1}{2}$  in. After a negative aspiration test 10 ml of solution is injected between the first rib and the skin. The needle is reintroduced on to the first rib 1 cm laterally to the first position and 10 ml similarly deposited. Third and fourth injections are made each 1 cm lateral to the last and at each point 10 ml is deposited between skin and rib.

Analgesia is rapid in onset if paræsthesias are present. If not an interval of 20 minutes may be necessary. A feeling of warmth and pins and needles precedes analgesia while motor paralysis when it occurs follows analgesia.

An area of skin over the point of the shoulder and another on the inner aspect of the upper arm from the axilla to its midpoint (intercostohumeral T 2) are not made insensitive. A subcutaneous band of injection downwards from the acromio-clavicular joint and surrounding the shoulder will render these areas analgesic.

Eucaine or metycaine 2 per cent solution will produce sensory and motor paralysis. Solution of 1 per cent will give sensory loss alone for about 1 hour. Adrenaline should be added to all solutions so that their final strength is about 1:300,000. For prolonged operation amethocaine or nupercaine 1:1000 is preferred. Lignocaine 1 to 2 per cent is excellent. Nerve suturing or trimming requires the use of the stronger solutions. Toxic and ill or feeble patients should have the strength of solution and not the volume reduced.

*Horner's syndrome* may or may not follow injection. It is due to paralysis of the cervical sympathetic chain and is characterized by (1) Small pupil (2) Ptosis (3) Enophthalmos.

It was described by Horner a Swiss ophthalmologist in 1869 having been previously noted by Claude Bernard in 1862 and by François Pourfais du Petit in 1727. The constriction gives place to dilatation in the darkness while the narrowing of the palpebral fissure is partly due to raising of the lower lid. Enophthalmosis not well marked in man. In this condition the eye becomes temporarily myopic.

Macintosh and Mushin point out three other signs of sympathetic paralysis —

- 1 Vasodilatation of nasal mucosa with engorgement and nasal obstruction (Guttman's sign)
- 2 Absence of sweating
- 3 Flushing of skin and conjunctiva

**COMPLICATIONS —**

- 1 Paralysis of phrenic nerve often occurs. At the level of the first rib the phrenic nerve is separated from the brachial plexus by the scalenus anticus muscle. Higher in the neck it is in the same fascial compartment as the upper

Another method of blocking the finger is to deposit 5-7 ml of 1 per cent lignocaine solution in the interosseous spaces at each side of it entering from the dorsal aspect and carrying the needle almost to the palmar skin. A similar technique—using less solution—can be employed on the toe.

## WRIST BLOCK

Circular lines of intradermal and subcutaneous infiltration are carried out just above the wrist joint.

**MEDIAN NERVE**—The median nerve at the wrist lies deeply between the flexor carpi radialis and the palmaris longus or in the absence of the latter it is medial to the flexor carpi radialis. It is injected with 5 ml of 1 per cent lignocaine immediately lateral to the tendon of the palmaris longus. Attempts to elicit paraesthesia are made but if unsuccessful the solution is injected nevertheless.

**ULNAR NERVE** divides two inches above the wrist joint into volar and dorsal branches.

The volar branch lies between the flexor carpi ulnaris tendon and the ulnar artery. The pisiform bone is immediately medial to it. It is blocked from a wheal immediately lateral to the flexor carpi ulnaris.

The dorsal branch is anesthetized by intradermal and subcutaneous injection along a line at the level of the ulnar styloid from the medial side of the tendon of the flexor carpi ulnaris to the middle of the back of the wrist.

**MUSCULOSPIRAL OR RADIAL NERVE** is the sensory nerve of the back of the lateral part of the hand. It can be blocked by infiltrating between the skin and the bone on the postero-lateral aspect of the wrist joint near the base of the thumb lateral to the radial artery.

Wrist block with a finger tourniquet is useful for surgery of the hand. For analgesia of the palm, web and dorsum of hand good results can be obtained by—

1. A median nerve block
2. An ulnar nerve block
3. A subcutaneous zone of analgesia (5 ml) across dorsum of wrist to block branches of the radial nerve.

## ELBOW BLOCK

Intradermal and subcutaneous circles of infiltration are made just proximal to the internal epicondyle.

**MEDIAN BLOCK** is obtained by injecting through a wheal placed midway between the outer side of the tendon of the biceps and the internal epicondyle or from a wheal 1 cm medial to the brachial artery at the bend of the elbow. The needle should be inserted in an upward direction. If paraesthesia is felt so much the better. 5 ml of 1 per cent lignocaine are used.

**MUSCULOSPIRAL BLOCK** is performed through a wheal 1 cm lateral to the tendon of the biceps at the line of the bend of the elbow. Alternatively a wheal is raised four finger breadths proximal to the lateral epicondyle of the humerus which overlies the point at which the nerve pierces the intermuscular septum.

**Brachial Plexus Block—Technique continued**

of local analgesic solution are now injected. The needle must not be too posterior on the rib in the substance or even behind the scalenus medius muscle. Paræsthesiæ are not elicited.

**KNIGHT'S THREE NEEDLE METHOD**—Raise a wheal a finger breadth above a point at the junction of the inner and middle thirds of the clavicle. A needle is passed through this wheal down to the upper surface of the first rib. A second needle is inserted parallel to the first midway between it and the clavicle so that its point makes contact also with the rib. The third needle is thrust in parallel to the others a finger breadth behind the first one on to the first rib and 10 ml of solution is injected through each needle as it is gradually withdrawn.

**Pitkin's Lateral (Paravertebral) Method\***—The fifth sixth seventh and eighth cervical and first thoracic nerves are blocked by the lateral approach in a similar manner to the technique adopted for block of the deep part of the cervical plexus. The patient lies on his back with his head rotated to the opposite side and with the shoulder depressed. A wheal is raised about 1½ in above the midpoint of the clavicle approximately on a level with the seventh cervical vertebra. Through this wheal a 2½-in needle is inserted towards the posterior tubercle of the transverse process of the seventh vertebra and when bone is contacted and an aspiration test proved negative 5–10 ml of local analgesic solution is injected. The needle is withdrawn almost to the skin and re-directed upwards to the transverse process of the sixth cervical vertebra and another similar injection made. Once again the needle is withdrawn almost to the skin and re-directed downwards towards the transverse process of the first thoracic vertebra and again after aspiration 5–10 ml are injected. It is important that the posterior tubercles are contacted as the anterior tubercles are not in relationship to the spinal nerves. The first injection blocks the fifth and sixth cervical nerves the second the seventh the last, the eighth cervical and first thoracic.

Brachial plexus block is a most satisfactory method of analgesia. For dislocations of the shoulder or elbow joint for tendon suture for manipulation of fractures under the X ray screen for suturing of lacerations etc. the method is excellent. A tourniquet may be applied to the upper arm even in the absence of analgesia of the inner aspect of the upper arm. It is more successful in cases with a previously painless limb than in those with an existing painful lesion e.g. a fracture or abscess.

**FIELD BLOCK FOR OPERATION ON THE FINGERS**

With a fine needle an intradermal wheal is raised on the dorsum of the finger near its base. From 5 ml to 10 ml of solution is injected into the substance of the finger through this wheal between the bone and the skin. Analgesia may take fifteen minutes to become established. The palmar skin is not pierced. Adrenaline should not be used.

allows easy retraction of the scapula. Diffusion of solution results in vagus block and so the reflexes usually associated with periosteal stripping are prevented.

Some advantages of regional analgesia over general anaesthesia for thoracoplasty: (1) Less haemorrhage (2) Safe for diathermy (3) Minimal respiratory—especially paradoxical—movements (4) Co-operation of patient especially regarding cough (5) Less upsetting to patients.

### PARAVERTEBRAL SOMATIC BLOCK

This method was introduced by Sellheim in 1909 and developed by Lawen in 1911 who called it paravertebral conduction anaesthesia. It involves injecting a local analgesic close to the vertebral column where the nerve trunks emerge from the intervertebral foramina.

Macintosh and Bryce Smith in an excellent monograph\* describe the paravertebral space as a wedge shaped compartment bounded above and below by the heads and necks of adjoining ribs posteriorly by the superior costotransverse ligament medially by the body of the vertebra and the intervertebral foramen and its contents laterally its apex leads into the ~~extra-~~ <sup>int-</sup>dural space.

There is no direct communication between one paravertebral space and another but an indirect communication exists medially through the intervertebral foramen with the extradural space. Spread from one paravertebral space to another across the extradural space is frequent and may involve nerves on the same or on the opposite side of the body.

When the first dorsal to second lumbar nerve roots are blocked their rami communicantes are blocked too.

Paravertebral cervical block is described under cervical plexus block.

**Paravertebral Thoracic Block.**—It is indicated for operations on the chest or abdominal wall diagnosis of cardiac pain from pain of skeletal origin for relief of post operative post herpetic pain and the pain of fractured ribs. Each thoracic nerve emerges from the intervertebral foramen and divides into anterior and posterior primary divisions. The latter supplies the skin and muscles of the back in their upper portion the former after giving off a ramus communicans comes to lie midway between the transverse processes. It is important to fix a fixed landmark the spine of the twelfth dorsal vertebra being a useful one. The last rib which makes an angle of 45° with the spines of the vertebra is traced out a perpendicular dropped from this rib to the middle line and measuring 5 cm. will strike the spine of the twelfth dorsal vertebra. Other fixed points are (1) The vertebra prominens—C7 (2) The spines of the scapulae with patient sitting upright—T3 (3) The angles of scapulae with patient sitting upright—T9 or 10. In the cervical and lumbar regions the spinous process is on a level with the transverse process of the same vertebra. In the mid and lower thoracic regions owing to the downward slope of

\* Macintosh, R. R. and Bryce Smith, R. *Local Anaesthesia Abdominal Surgery* 1953. Edinburgh and London: E. and S. Livingstone.



**Elbow Block—Musculospiral Block** *continued*

and is close to the bone. The needle is advanced perpendicularly to the skin towards the bone and 20 ml of analgesic solution is deposited above and below the point of injection.

**ULNAR BLOCK** is performed where the nerve can be palpated behind the internal epicondyle.

### **REGIONAL ANALGESIA FOR THORACOPLASTY (MAGILL SEMB)**

Thoracoplasty was first performed in Britain by Morrison Davies in 1912.

Premedication for operations done under regional analgesia should be as follows: phenobarbitone 3 gr one and a half hours before operation—by mouth; omnopon  $\frac{1}{4}$  gr one hour before with additional dose a half hour before if necessary. If cough is active infection may be spread.

The technique requires two solutions: one containing procaine 0.5 per cent and amethocaine hydrochloride 1-1000 in normal saline; the other containing procaine 0.25 per cent and amethocaine hydrochloride 1-2000 in normal saline. Adrenaline is added. Many workers are now using instead 0.4 per cent and 0.2 per cent xylocaine solutions with very good results.

The following injections are made—

- 1 Supraclavicular brachial plexus block using the stronger solution
- 2 Posterior intercostal nerve block of the upper five or six nerves at points  $1\frac{1}{2}$  in from the midline using the stronger solution 5-10 ml for each nerve
- 3 Infiltration of the line of incision intradermally and subcutaneously using the weaker solution
- 4 Injection of 20-30 ml of weaker solution under the scapula
- 5 Injection into areas to receive towel clips
- 6 A wheal is raised 1 in. from the sternum in the first five intercostal spaces. This blocks any fibres which may come from the other side overlapping the middle line of the body.

Steps 1 and 6 are sometimes omitted while the posterior intercostal block can be performed by the surgeon under direct vision if necessary.

The second stage operation requires a less extensive block. About 70 ml of stronger and 150-200 ml of weaker solution may be used.

Scurr has modified this technique as follows\*. He uses xylocaine 0.4 per cent for nerve block and 0.2 per cent for infiltration. With this drug he finds that analgesia lasts for four hours, is rapid in onset and associated with low toxicity. The solution has excellent penetrating and spreading powers, gives intense analgesia together with block of tactile sensations. He blocks the brachial plexus by the lateral or paravertebral approach thereby paralysing the nerve to the serratus anticus which

The needle inserted through the wheal opposite the first lumbar spine blocks the twelfth dorsal nerve

Successful paravertebral injection produces visceral (i.e. splanchnic) block as well as analgesia of the abdominal wall. This is because the white rami are bathed in solution. Injection can be given with the patient in the lateral sitting or prone positions—the last with a pillow under the abdomen.

Operations on the following regions can be performed by paravertebral block—

Appendix T 10-L 2

Hernia, T 11-L 2

Upper abdomen T 5-12

Lower abdomen T 7-L 3

The method is very useful for cases of strangulated hernia. The following organs receive their visceral nerve supply as follows

Kidney T 10 to L 1 Ureter T 11 to L 1 Testis T 10

Epididymis T 11-12 Bladder T 11-12 L 1 and S 3-4

Prostate T 10-11 S 1 to 5 Ovary T 10 Cervix uteri

T 11-12 and S 1 to 4 Uterine body T 10 to L 1

Nerve supply of suprarenal glands is from the three splanchnic nerves and first and second lumbar ganglia via coeliac inferior phrenic and renal plexuses. Twigs also received from vagus. Chief root value—T 10-L 2

## THORACIC SYMPATHETIC BLOCK

Block of the thoracic ganglia of the sympathetic chain by paravertebral injection of local analgesic drugs. First carried out by Sellheim (1905) and Kappis (1900). Alcohol first used to cause a prolonged block by Swetlow in 1926.

**Anatomy**—The thoracic portion of the sympathetic chain usually consists of eleven ganglia which with the exception of the last two lie against the heads of the ribs and are covered by the costal pleura. The last two are placed on the sides of the bodies of the eleventh and twelfth thoracic vertebrae. Two rami communicantes, a white and a grey, connect each ganglion with its corresponding spinal nerve (the cardiac afferents travel with the thoracic cardiac sympathetic fibres; enter the upper four thoracic sympathetic ganglia and without synapsing there go with the corresponding white rami to the mixed sternal nerves and the posterior roots). Stellate ganglion block will interrupt conduction along fibres from the upper four thoracic ganglia.

**Site of Injection for Various Conditions**—Anatomists differ in their recommendations. Mandl in his book *Paravertebral Block\** (1947) recommends the following: Angina pectoris T 1-4 both sides. Bronchial asthma T 1-5 both sides. Paroxysmal tachycardia T 1-2 both sides. Biliary colic T 6-10. Renal colic T 12-L 2. Uterine contractions (labour pains) T 11-12. Pancreatic pain (acute pancreatitis) T 8-10 on left side. Upper limb T 2 or T 2 and 3.

**Paravertebral Somatic Block, continued**

the spinous processes the tip of each spinous process corresponds to the transverse process of the vertebra below thus an injection at the level of the ninth thoracic spinous process will block the tenth nerve

**TECHNIQUE 1**—Wheals are raised opposite the lower borders of the dorsal spines two finger breadths from the middle line. Through each wheal a needle is thrust perpendicular to the skin, until it strikes bone which is near the lateral extremity of the transverse process at a depth from the skin of 1 to 2 inches. The needle is partially withdrawn and directed upwards over the upper border of the transverse process but not deep to it. At this point 5-10 ml. of solution is injected. Xylocaine 1 to 1.5 per cent, procaine 1 per cent, or amethocaine 1-1000 or nupercaine 1-1500 is suitable. It is important to avoid puncturing the dura or a vessel aspiration tests should be made.

**TECHNIQUE 2**—Wheals are raised three finger breadths from the middle line. Through each a 10-cm. needle is inserted making an angle of 45° with the sagittal plane until it glances off the lower border of the rib or passes between the ribs. It is slowly advanced until at a depth of 6-7 cm. it strikes the body of a vertebra in the paravertebral space. 10 ml. of solution are now deposited between the rib and the vertebra taking care not to puncture the dura. Molesworth recommends the use of a sterile protractor to measure the angle. To avoid unnecessary needle punctures Macintosh and Bryce-Smith suggest that only alternate nerves need be injected using 20 ml. of solution and relying on overflow via the extradural space to cause block of the un.injected nerves.

Puncture of the pleura or lung is not a serious error in technique although it may lead to waste of solution and bad results. Analgesia stops 1 in. from the midline owing to overlap from the other side. Thus in unilateral blocks a line of subcutaneous infiltration along the midline must be made.

**Paravertebral Lumbar Block.**—As the upper edge of the spinous process of a lumbar vertebra is level with the transverse process of that vertebra, wheals are raised opposite this upper edge two finger breadths from the middle line. Through each wheal a needle is introduced at right angles to the skin surface until its point touches the long transverse process at a depth of 4-5 cm. A piece of rubber threaded on the needle as a marker is now set 3 cm. from the skin. The needle is partially withdrawn and directed slightly upwards and inwards so that it glances off the upper border of the transverse process. It is advanced a further 3 cm. i.e. to the marker and 2-10 ml. of solution are injected after making an aspiration test. The needle is moved to and fro slightly during the injection. Care is necessary to avoid intrathecal injection.

The fifth nerve is injected through the same wheal as the fourth, only the needle slides off the lower border of the transverse process of the fifth vertebra, instead of over its upper border.

The needle inserted through the wheal opposite the first lumbar spine blocks the twelfth dorsal nerve

Successful paravertebral injection produces visceral (i.e. splanchnic) block as well as analgesia of the abdominal wall. This is because the white rami are bathed in solution. Injection can be given with the patient in the lateral sitting or prone positions—the last with a pillow under the abdomen.

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**Thoracic Sympathetic Block continued**

**Technique** —The posterior approach is the only one possible. Patient should be either sitting with head flexed or in the lateral spinal position. Pneumothorax is a very likely complication.

A wheal is made two finger breadths from the midline over the transverse process of the desired vertebra. In the upper thoracic region owing to the obliquity of the spinous processes the spine of a given vertebra is level with the transverse process of the vertebra below. Through the wheal a 10 cm needle is introduced at right angles to the skin surface and contact is made with the transverse process and then slipped inferior to it. The marker is set at 4 cm to the skin surface the needle inclined 20° to the median sagittal plane and the needle is advanced until it makes contact with the body of the vertebra at a depth of about 3 cm. Into each ganglion 5 ml of 2 per cent procaine is injected after it has been ascertained that the needle point is not intravascular, intrapleural or intrathecal.

**Pulmonary Plexus Block** —The pulmonary plexus is a network of autonomic fibres akin to the celiac plexus lying anterior to the bodies of the upper thoracic vertebrae. Block is carried out similarly to thoracic sympathetic ganglionic block but needle is advanced a further 1 cm so that solution of analgesic drug comes to lie in the vicinity of the plexus. It is said to be beneficial in cases of very severe bronchospasm.

**LUMBAR SYMPATHETIC BLOCK**

**Anatomy** —The sympathetic trunk in the lumbar region consists of four ganglia and their interconnecting fibres. It lies on the anterior aspect of the bodies of the lumbar vertebrae immediately medial to the psoas muscle which fills the triangular space between the vertebral bodies and the transverse processes. A tendinous arch which gives part origin to the psoas muscle connects the upper and lower borders of each lumbar vertebra and forms a tunnel around the side of the bone in which the lumbar vessels and the great ramus communicans runs. The fatty tissue occupying this tunnel is an extension of that in the extradural space and it passes through the intravertebral foramina as far forward as the sympathetic chain (Bryce Smith) \*

Disturbances of function of the sympathetic nervous system can produce (1) Vasospasm (2) Pain (3) Visceral dysfunction. Sympathetic block can remedy all of these either temporarily for diagnosis or permanently by interrupting a vicious circle.

**Indications** —

1. Peripheral arterial disease. This may be (a) Vasospastic (Raynaud) (b) Vasospastic and organic (Buerger) (c) Degenerative organic (arteriosclerosis). Increased blood supply to the limb is shown by (i) Increased surface temperature (ii) Increased oscillations shown by an oscillogram (iii) Increased function e.g. later onset of claudication.

- 2 Traumatic vasospasm so-called local shock.
- 3 Post traumatic and post infection edema.
- 4 Acute arterial occlusion
- 5 Thrombophlebitis the symptoms of which are due to arterial spasm in walls of veins
- 6 Delayed healing of fractures
- 7 Trophic ulcer Sudeck's atrophy traumatic osteoporosis and Charcot's joint
- 8 Causalgic states following trauma
- 9 Blockade of efferent and afferent sympathetic fibres from pelvic organs
- 10 Abolition of rhythmic labour pains due to uterine contractions

Sympathetic block when correctly done gives more complete release of vasomotor tone than do the chemical ganglionic blocking agents

#### Technique —

**POSTERIOR APPROACH**—The patient is placed in the prone position with two pillows flexing the lumbar spine or in lateral spinal position with the affected side uppermost and the spine flexed. He should be premedicated with a barbiturate. The procedure can be carried out in the patient's bed if necessary.

Skin wheals are raised at points 5 cm lateral to the upper borders of the spinous processes of the 2nd, 3rd and 4th lumbar vertebrae. Successful injection at these points blocks all the vasoconstrictor impulses to the lower limb. These points lie immediately above the transverse processes of the corresponding vertebrae. A spinal needle is introduced through each wheal at right angles to the skin for 4-5 cm and should encounter the transverse process. It is slightly withdrawn and directed upwards so that it passes between the transverse processes. It is also directed slightly inwards. After travelling 3-4 cm from the transverse process the needle should make contact with the anterolateral aspect of the body of the vertebra. After careful aspiration to exclude both blood and cerebrospinal fluid 20 ml of 1 per cent lignocaine are injected at each site. If force is required needle tip is in anterior vertebral ligament or the psoas muscle and should be slightly withdrawn. It spreads out in the retroperitoneal tissue. The spinal lumbar nerves run midway between the spinous processes so if the needle point is kept in relation to the upper border of the transverse process pain from hitting a nerve should be avoided. The lumbar arteries—branches of the aorta—with their veins must also be avoided. After 5 to 20 minutes the leg becomes less painful its temperature increases and it becomes dry its superficial veins dilate and there is hyposensitivity to pin prick.

See also good article by R. Bryce Smith\* in which he recommends the insertion of a 12-cm needle at an angle of 70° through a wheal three finger breadths lateral to the superior point

**Lumbar Sympathetic Block—Technique continued**

of the spinous process of L 3. It should miss the transverse process and come into contact with the body of the vertebra in the psoas tunnel. 15–20 ml of analgesic fluid is now injected.

**LATERAL APPROACH \***—The patient is placed in the lateral position. A wheal is raised at the apex of the lumbar triangle (boundaries: lower border of last rib, superior border of iliac crest, lateral border of paravertebral muscle group). This point is at the level of L 2. A 6 in. needle is inserted through the wheal and angulated 15° anteriorly and thrust in until bone is struck; this should be the lateral aspect of the body of the second lumbar vertebra. At this point 10 ml of solution is deposited. Withdrawal is made and the needle angulated a further 15° anteriorly and again bone is struck and more solution injected. A third withdrawal and insertion at an additional 15° angle anteriorly is made and a third injection given. Thus the anterolateral aspect of the body of the second lumbar vertebra is well surrounded with solution.

Chemical sympathectomy should not be done in this way for fear of involving the third lumbar nerve as it leaves the intervertebral foramen.

Low caudal block produces the same results but is not unilateral; it produces in addition motor paresis and analgesia which may be useful objective signs of successful sympathetic paralysis. The needle can be left in the sacral canal so that analgesia lasting for 4 hours can be produced. After a rest a similar period of analgesia can be instituted.

For chemical sympathectomy Haxton† recommends the injection of 6 per cent phenol in water. He uses 2–3 ml for each ganglion after injecting 2 ml of 4 per cent procaine which verifies the position of the needle. He uses 12 cm needles and advances them from wheals 7 cm from the midline.

**FIELD BLOCK FOR MASTECTOMY**

**Anatomy**—The overlying skin of the breast is supplied by the anterior and lateral branches of the 4th, 5th and 6th intercostal nerves.

**Technique**—For non radical mastectomy wheals are raised a finger breadth from the circumference of the breast—about eight in number. Through each wheal an injection is made towards the nipple so that solution is deposited between the breast substance and the pectoral muscles. Injection should not be made into the breast substance. Finally the circumferential wheals are joined by intradermal and subcutaneous injections.

For radical mastectomy a much more extensive procedure is required. The solution should contain one of the longer acting drugs such as amethocaine or nupercaine. The following procedures are necessary —

Wallace, G., *Anæsthesia*, 1955, 16, 234.

† Haxton, H. A. *Brit. med. J.*, 1949, 1, 27.

- 1 Brachial plexus block
  - 2 Block of the first to tenth intercostal nerves either adjacent to the vertebral column or at the posterior axillary fold
  - 3 Block of the descending branches of the cervical plexus by infiltrating along acromion clavicle and sternum
  - 4 Infiltration of line of incision intradermally and subcutaneously
- Induction of local analgesia for radical mastectomy is thus seen to be almost a major operation in itself. It is seldom indicated

## INTERCOSTAL NERVE BLOCKS

### The Cutaneous Nerves of the Trunk.—

#### ANTERIORLY —

- a The supra acromial supraclavicular and suprasternal branches of the superficial division of the cervical plexus (C 3-4)
- b The anterior rami of the thoracic nerves excluding T 1
- c The iliohypogastric and ilio-inguinal nerves (L 1)

POSTERIORLY — The posterior rami of C 2 3 4 5 T 1 to 12 L 1 to 3 the five sacral and the coccygeal nerves

**Anatomy of Spinal Nerves (Figs 52 53) —** Each nerve is formed by the union of the anterior (motor) and the posterior (sensory) root the latter has a ganglion on it. The mixed spinal nerve soon divides into anterior and posterior primary divisions (rami). The thoracic or dorsal nerves then are distributed as follows —

The posterior primary divisions are smaller than the anterior. They turn backwards and divide into medial and lateral branches (except C 1 S 4 and S 5 coccygeal) which supply the muscles and skin of the back. Above the 6th dorsal spine the sensory nerves come from the medial branches and emerge  $1\frac{1}{2}$  finger breadths from the middle line the lateral branches supplying the muscles below the 6th dorsal spine the lateral branches supply the skin emerging a hand breadth from the middle line the medial branches here supplying the muscles. The first sixth seventh and eighth cervical and the fourth and fifth lumbar posterior primary divisions give no cutaneous branches.

The anterior primary divisions in the thoracic region of the second to sixth nerve are each connected to the lateral sympathetic chain by a grey and a white ramus communicans each crosses the paravertebral space between the necks of contiguous ribs and then enters the subcostal groove where it lies below the vein and artery and between the internal and external intercostal muscles as far as the anterior axillary line at which point the nerves come into direct relationship with the pleura as the internal intercostal muscle terminates. It supplies muscular branches to the intercostal muscles and supplies lateral and anterior cutaneous branches to supply the skin of the chest and abdomen. The seventh to eleventh nerves pass below and behind the costal cartilages between the slips of the diaphragm running between the internal oblique and transversus muscles to enter the posterior layer of the rectus sheath. They run deep to the rectus pierce and supply it and end as anterior cutaneous nerves.





The lateral cutaneous branch emerges in the mid axillary line and divides into anterior and posterior branches which supply the skin on the lateral wall of the chest as far forward as the nipple line.

The anterior cutaneous branch is the termination of the intercostal nerve. It supplies the skin on the front of the chest, internal to the nipple line.

**EXCEPTION**—The first nerve gives neither lateral nor anterior cutaneous branches; the skin over the first intercostal space being supplied by the descending branches of the cervical plexus (C 3-4). The lateral cutaneous branch of the second intercostal nerve crosses the axilla and becomes the intercostobrachial nerve supplying the skin on the medial aspect of the arm. The lateral cutaneous branch of the twelfth dorsal nerve, which does not divide into anterior and posterior branches, crosses the iliac crest to supply the skin of the upper part of the buttock as far as the great trochanter. The twelve thoracic and first lumbar nerves supply sensory branches to the anterior chest and anterior abdominal wall, the parietal pleura and the parietal peritoneum.

The tenth nerve—lateral and anterior cutaneous branches—supplies the area of the umbilicus.

The ninth, eighth and seventh supply the skin between the umbilicus and the xiphisternum.

The eleventh and twelfth and first lumbar nerves supply the skin between the umbilicus and the pubes.

#### **Technique of Intercostal Nerve block —**

1. **AT THE ANGLE OF THE RIB** (Sellheim 1906, N. R. James 1943) —At this point the nerve becomes relatively superficial, lateral to the erector spinae muscle. In this technique the rami communicantes conveying afferent impulses are not blocked, so splanchnic analgesia is necessary in addition. The patient is arranged in the lateral spinal position with his back well arched over the edge of the table. After swabbing with antiseptic and fixing sterile towels, two lines are drawn, one on each side, four finger breadths from the middle line, with an iodine swab. The lines should extend from the spines of the scapulae to the iliac crests. At a point where the lower border of the 12th rib on the patient's upper side crosses the iodine line, a needle is introduced (through an intradermal wheal if necessary) until it makes contact with the rib. It is then partially withdrawn and advanced until it slips past the lower border of the rib for one-eighth of an inch. 10 ml. of analgesic solution is then injected while the needle point is slightly advanced and withdrawn so as to surround the nerve with analgesic solution. The intercostal nerve is thus surrounded by a zone of solution as it lies in the subcostal groove between the internal and external intercostal muscles. The needle should be mounted on a syringe so that should the pleura be punctured, no air will enter the pleural cavity. The needle is then withdrawn until its point is just beneath the skin, so that it can act as a marker. The eleventh to the sixth nerves are now injected on the upper side, followed by the lower seven nerves on the patient's

**Technique of Intercostal Nerve block continued**

more dependent side. Before the sixth and seventh nerves can be injected the patient's scapulæ must be drawn laterally by crossing his arms over his chest. The needle pierces the trapezius, the latissimus dorsi and the two intercostal muscles.

Posterior splanchnic block can be performed with the patient in the same position.

2. **IN THE POSTERIOR AXILLARY LINE**—Similar injections can be carried out with the patient supine and with his arms abducted to a right angle. In this position the ribs and so the intercostal nerves are not so deeply placed. A block in the mid axillary line muscels the lateral cutaneous nerve.

Blocking of the lower seven intercostal nerves on each side results in analgesia of the anterior abdominal wall from just below the nipple line to a point midway between the line joining the anterior superior iliac spines and the pubic bone. In addition it produces analgesia of the parietal peritoneum and relaxation of the muscles of the anterior abdominal wall. It is especially useful when scars from previous operations disturb the relations of the abdominal wall. For analgesia of the viscera splanchnic analgesia is required in addition.

### **INTERCOSTAL BLOCK FOR RIB RESECTION IN DRAINAGE OF EMPYEMA**

The surgeon is asked to mark out the position of the incision he wishes to make and it is infiltrated intradermally and subcutaneously with  $\frac{1}{2}$ –1 per cent lignocaine solution. An intradermal and subcutaneous line of infiltration is carried out one rib above and one below the length of rib to be removed. These lines extend 1 in. in front and 1 in. behind the proposed incision and these extremities are joined by intradermal and subcutaneous infiltrations so that a rectangle is marked out. The intercostal nerves within this rectangle are blocked at its posterior extremity with 2 per cent procaine solution. There may be slight discomfort during the stripping of the periosteum from the rib.

Local block is usually the preferred method of anæsthesia in these operations. If the patient is well enough he should sit side ways across the table to prevent him drowning in his own pus if the abscess ruptures into a bronchus. Otherwise he is placed in the lateral position with the head and shoulders elevated.

### **SPLANCHNIC ANALGESIA**

#### **Anatomy —**

**SEMI UNAR OR CÆLIAC GANGLIA**—Two in number one on each side of the midline lying on the aorta and the crura of the diaphragm just above the pancreas at the level of the first lumbar vertebra between the suprarenal glands and behind the stomach and lesser sac. The renal vessels are inferior to the ganglia while the vessels to the suprarenals often pass through the ganglion. They are connected with each other and with their associated ganglia (superior mesenteric and inferior mesenteric etc.) by a network of nerve fibres around the cœliac axis artery. These fibres are post ganglionic fibres of the

greater and lesser splanchnic nerves. From this mass of retro-peritoneal nerve tissue fibres pass with the arteries to the abdominal viscera. These plexuses also receive twigs from the vagi and the phrenic nerves. The semilunar or celiac ganglia with the aortico-renal and superior mesenteric ganglia together make up the solar or epigastric plexus.

**GREATER SPLANCHNIC NERVE** (the superior thoracic splanchnic nerve) like the lesser and the least splanchnic is composed of pre-ganglionic fibres which are in effect elongated white rami. The majority of its fibres are myelinated. It rises from the union of four or five roots coming from the thoracic sympathetic ganglia which receive white rami from the fourth to the tenth dorsal nerves. The nerve enters the abdomen through the crus of the diaphragm on each side with the lesser and least splanchnic nerves and enters the corresponding semilunar ganglion. It also contains visceral afferent fibres. Within the abdomen the nerve lies between the diaphragm and the suprarenal gland on each side.

**LESSER SPLANCHNIC NERVE** (the middle thoracic splanchnic nerve) —This arises from the lower thoracic ganglia of the sympathetic cord connected with the tenth and eleventh dorsal nerves. It enters the corresponding semilunar ganglion.

**LEAST SPLANCHNIC NERVE** (the inferior thoracic splanchnic nerve) —This arises from the last dorsal ganglion and enters the renal plexus and the posterior renal ganglion.

Afferent fibres from the abdominal viscera both sympathetic and parasympathetic (vagus) pass through the celiac ganglia. Afferent fibres from the pelvic viscera travelling through the nervi erigentes (S 2 3 4) do not.

**THE LUMBAR SPLANCHNIC NERVES** —Are presumably blocked when the celiac plexus is blocked by spreading of solution. The first lumbar splanchnic nerve arises from the first lumbar ganglion, the second from the second and third ganglia, the third from the second, third and fourth ganglia, the fourth from the fourth and fifth ganglia. The last two join the superior hypogastric plexus.

**THE SUPERIOR HYPOGASTRIC PLEXUS** (or pre sacral nerve which is really pre lumbar) —Extends from the third lumbar vertebra to the first sacral where it ends by dividing into the right and left hypogastric nerves or plexuses. It lies in front of the lower part of the abdominal aorta behind the peritoneum.

**Afferent Pathways from Upper Abdominal Viscera** —These visceral afferents travel from sensory nerve-endings in the walls of the viscera mesentery etc. via the splanchnic nerves and enter cord with the white rami of the lower six thoracic nerves having their cell stations in the posterior root ganglia of these nerves.

**Nerve supply to Suprarenal Glands** —Pre ganglionic fibres do not synapse in the celiac or other pre aortic plexuses but pass directly to end around chromaffin cells of the medulla. In addition to the lesser splanchnic fibres go to the suprarenals from the tenth thoracic to the second lumbar nerves.

*Splanchnic Analgesia continued*

**Nerve supply to Testis, Kidney, and Ureter**—From each organ via perivascular nervous plexus to aortic and renal plexuses and into white rami and posterior nerve roots from T 10 to L 1

**Technique of Splanchnic Block**—Splanchnic block can be performed before laparotomy (Wendling) or after laparotomy from the front (Braun) or before laparotomy from behind (Kappis). The Braun technique is usually performed by the surgeon

1 **BRAUN'S METHOD**—Described in 1919. With the abdomen opened the liver is gently retracted upwards and the stomach is drawn to the left. The anterior aspect of the body of the first lumbar vertebra is located medial to the lesser curvature of the stomach. The aorta is retracted laterally and the long Braun needle is inserted down to the bone and 50 ml of solution injected.

2 **KAPPIS'S METHOD**—Described in 1914. The patient is in the spinal position sitting or lying prone. The fourth interspace is located lying on or below the intercostal line by counting upward the spine of the first lumbar vertebra is identified. Wheals are raised four finger breadths from this spine one on each side of the midline. The wheals must be below the twelfth rib.

A long needle is inserted at an angle of 45° to the median plane through this wheal with its bevel facing inwards. It is directed slightly upwards and thrust in until it makes contact with the body of the first lumbar vertebra. It is then partly withdrawn and its point directed more laterally until its bevel is felt to glance past the lateral aspect of the body of the vertebra. The needle is then advanced a further 1 cm and after a most careful aspiration test 50 ml of solution is injected. The average distance between the skin and the plexus is 7–10 cm. If blood is aspirated into the syringe the needle point may be in the vena cava or the aorta and must be moved until it is free of these vessels. Bilateral block is probably unnecessary.

The usual strengths of solution employed are procaine or lignocaine 0.5 per cent amethocaine 1–2000 to 1–4000 nupercaine 1–2000.

Splanchnic block causes a profound fall in blood pressure which can be partially controlled by ephedrine or methedrine should it be considered necessary. It produces analgesia of the abdominal viscera with the exception of the pelvic viscera i.e. the sigmoid colon, rectum, bladder and reproductive organs. The bowel becomes contracted and ribbon-like. The patient must be particularly well premedicated intravenous morphine being given until his mental state is calm. The surgeon must be light handed especially when the peritoneal cavity is being explored as its lateral walls are not rendered insensitive either by the splanchnic block or the abdominal field block.

Therapeutically splanchnic block is useful in the treatment of acute pancreatitis and also in fibrocystic disease of young infants perhaps because it relaxes the sphincter of C — 1<sup>st</sup> causes

a greater blood-supply to be diverted to the pancreas. The block may be repeated if desirable. It has also been used in the terminal stages of upper abdominal cancer to relieve pain.

**Abdominal Aortography**—This was pioneered by Dos Santos in 1929. The technique is as for Lappin's splanchnic block on the left side of the body. The patient is given a suitable sedative such as omnopon and scopolamine and in the X-ray room is given a test dose of 1 ml. of 70 per cent diodone. If no toxic reaction is seen the patient lies prone and each lumbar spine is marked. If aortography is being performed to show the iliac vessels the injection is made opposite L<sub>3</sub>. To display the renal vessels the injection is opposite L<sub>1</sub> while to display the coeliac axis and its branches the injection is made opposite T<sub>12</sub>. Just before the injection is given the patient may receive 100 mg. of pethidine intravenously. The injection needle should be 16 s.w.g. 7 in (18 cm.) in length. As this method of diagnosis has been known to lead to paraplegia spinal and extradural analgesia should be avoided. With the long needle proved by aspiration of arterial blood to be within the aorta 30 ml. of 70 per cent diodone are rapidly injected and radiographs quickly taken.

### ABDOMINAL FIELD BLOCK

**Anatomy**—The superficial fascia in the upper abdomen is a single fatty layer but from a point midway between the umbilicus and the pubis two layers can be distinguished the fascia of Scarpa and the superficial fascia of Camper.

Camper's fascia passes over the inguinal ligament and is continuous with the superficial fascia of the thigh. It is continued over the penis, spermatic cord and scrotum where it helps to form the dartos muscle. In the female it is continued into the labia majora.

Scarpa's fascia is tougher. It blends with the deep fascia of the thigh and like Camper's fascia is continued over the penis and helps to form the dartos. From the scrotum it becomes continuous with Colles's fascia over the perineum.

**EXTERNAL OBLIQUE**—The largest and most superficial of the muscles of the anterior abdominal wall. It arises from the external surfaces of the lower eight ribs and fans out to its attachments which are the anterior half of the outer lip of the iliac crest and the aponeurosis which commences on a line drawn from the ninth cartilage to the anterior superior iliac spine. The aponeurosis is attached below to the anterior superior spine and to the pubic tubercle. It thus forms the inguinal ligament. In the midline it forms the linea alba which runs from the symphysis pubis to the xiphisternum. The subcutaneous or external inguinal ring is an opening in the aponeurosis.

The fibres of the external oblique pass downwards and inwards like those of the external intercostal muscles.

**INTERNAL OBLIQUE**—This is a thinner layer than the above. It arises from (a) The lateral half of the inguinal ligament (b) The anterior two thirds of the iliac crest (c) The lumbo-dorsal fascia.

Abdominal Field Block—Anatomy *continued*

*Insertion* is as follows. Fibres arising from (a) arch over the spermatic cord or round ligament and together with fibres from the transversus abdominis form the conjoined tendon or falx inguinalis which is attached to the pubic bone. The fibres from (b) and (c) are inserted into an aponeurosis which divides at the lateral border of the rectus into two layers which partially invest this muscle and then fuse with the linea alba. Above the posterior layer is attached to the lower borders of the lower six ribs and their costal cartilages.

The fibres of this muscle run upwards and inwards.

TRANSVERSUS ABDOMINIS—*Arises*—

- a From the lateral third of the inguinal ligament
- b From the anterior three-quarters of the iliac crest
- c From the lumbodorsal fascia
- d From the inner surfaces of the lower six ribs and their cartilages interdigitating with the diaphragm

It is inserted into a broad aponeurosis the lower fibres forming part of the conjoined tendon and being inserted into the pubic bone. The remainder of the aponeurosis is inserted into the linea alba the upper three quarters passing behind the rectus the lower quarter passing in front of it. In the upper abdomen the aponeurosis is adherent to the peritoneum.

The fibres of the muscle run transversely. Between it and the external oblique run the lower intercostal iliohypogastric and ilio inguinal nerves.

RECTUS ABDOMINIS—Each muscle arises from the crest of the pubis and from the ligaments in front of the symphysis. Inserted into the anterior aspects of the fifth sixth and seventh costal cartilages and into the xiphisternum.

Three tendinous intersections cross the muscle and are firmly attached to the anterior layer of its sheath but not to the posterior layer. One is at the level of the xiphisternum one at the umbilicus and the third one midway between. They represent prolongation of the 8th 9th and 10th ribs and are in relationship to the corresponding intercostal nerves.

The rectus sheath is not arranged in the same way throughout its length and is described in three sections—

- 1 From the costal margin to a point midway between the umbilicus and the pubis. Here at the outer border of the muscle the aponeurosis of the internal oblique divides into anterior and posterior layers the former fuses with the aponeurosis of the external oblique and passes in front of the rectus the latter fuses with the aponeurosis of the transversus abdominis and passes behind the rectus. Just below the costal margin muscular fibres of the transversus abdominis pass almost to the midline and unless this muscle is well relaxed the peritoneum in this situation cannot easily be sutured. Where the posterior sheath ends i.e. between the umbilicus and the pubis forms

an arched fold, the semicircular line of Douglas. At this point the inferior epigastric artery enters the rectus sheath.

- 2 From the semicircular line to the pubis all of the aponeurosis passes anterior to the rectus. The fascia transversalis lies posterior to the muscle and with the extraperitoneal fat separates it from the peritoneum.
- 3 Above the costal margin the anterior wall of the sheath is formed exclusively from the aponeurosis of the external oblique. Posteriorly the rectus is in relation to the costal cartilages of the 5th, 6th and 7th ribs.

The rectus sheath contains in addition to the rectus and pyramidalis muscles the superior and inferior epigastric vessels and the terminations of the lower six intercostal nerves and vessels. The nerves pierce the lateral margin of the sheath and run in relation to its posterior wall before they enter the substance of the muscle.

**PYRAMIDALIS**—A small muscle on each side within the rectus sheath arising from the pubis and inserted into the linea alba. It is well developed in marsupial mammals and serves to strengthen the linea alba.

The abdominal muscles are supplied by the lower six intercostal nerves and by the iliohypogastric and ilioinguinal nerves.

They are accessory muscles of expiration and help to compress the abdominal viscera as in defecation etc.

**TRANSVERSALIS FASCIA**—A thin membrane continuous with the iliac and pelvic fascia. In the inguinal region it is stronger and thicker than elsewhere and through it at the abdominal inguinal (internal inguinal) ring passes the spermatic cord or the round ligament.

**SENSORY NERVE SUPPLY OF ABDOMINAL WALL**—The skin in the region of the nipple is supplied by the fifth dorsal nerve.

Skin in the epigastrium is supplied by the seventh nerve.

Skin in the region of the umbilicus is supplied by the tenth nerve.

Skin midway between the umbilicus and the pubis is supplied by the twelfth nerve.

Skin of the hypogastrium is supplied by the iliohypogastric nerve (L 1).

Intercostal nerves and the last dorsal nerve pass under the costal margin between the slips of the diaphragm and run forwards between the internal oblique and the transversus abdominis before they pierce the lateral margin of the rectus sheath. After lying behind the rectus muscle they pierce its substance and supply it and end as anterior cutaneous nerves.

**Technique of Abdominal Field Block**—This should be commenced 15–20 minutes before the incision is to be made.

Wheels are raised—

- 1 At the tip of the xiphisternum

- 2 One on each side at the 9th costal cartilage where the rectus muscle crosses it



Technique of Abdominal Field Block *continued*

- 3 One on each side at the lateral margin of the rectus just above the umbilicus
- 4 One on each side at lateral margin of rectus below umbilicus —if the incision is to be prolonged

Through wheals 2 3 and 4 a needle is inserted perpendicularly until it meets the resistance of the rectus sheath. If the patient is conscious he will experience pain when the anterior layer of the rectus sheath is pierced. The needle is advanced a further  $\frac{1}{2}$  cm and 5 ml of solution is injected into the sheath. After withdrawal into the subcutaneous tissue the needle is inclined upwards and downwards so that more solution is deposited into the rectus sheath. It is important to remember the positions of the tendinous intersection so that solution is deposited between each pair to ensure even distribution of the analgesic drug. After completion of the deep injections the wheals are joined together along the lateral margin of the rectus by lines of subcutaneous injection. Similarly wheal 1 is joined to each wheal 2 along the costal margin. A total of 100–200 ml of solution is used.

A costo iliac block gives a wider zone of analgesia and relaxation than the rectus sheath block outlined above but is more difficult to carry out successfully. Wheals are raised on each side along the costal margin and vertically downward to the iliac crest. Solution is deposited from needles passed through these wheals into the subcutaneous and muscular layers of the abdominal wall remembering that laterally the intercostal nerves lie between the transversalis and internal oblique. The wheals are joined together by subcutaneous infiltration as described for rectus sheath block. In muscular subjects in addition solution can be injected into the rectus sheath. The volume of solution required is 150–200 ml.

Rectus sheath block (Carl Ludwig Schleich 1899) is an excellent method of producing muscular relaxation when combined with a light general anæsthetic and if the incision is to be midline or paramedian it is usual to do both sides. Perforation of the peritoneum should be avoided but in the absence of peritonitis or adhesions no serious harm is likely to result. The anterior layer of the rectus sheath is detected by the needle throughout its whole extent but the posterior layer only for about 3 in above and below the umbilicus. Solution is placed posterior to the muscle so that the intercostal nerves supplying it together with the zone of skin medial to its outer border are blocked. If the abdomen shows the scar of a previous operation abdominal field block may be difficult and undesirable and intercostal block or paravertebral block may be indicated if the operation is to be performed under local analgesia.

Abdominal field block renders the abdominal wall and its underlying parietal peritoneum insensitive. To block pain impulses from the viscera and posterolateral parietal peritoneum either light general anæsthesia or a splanchnic block is required.

## FIELD BLOCK FOR REPAIR OF INGUINAL HERNIA

**Anatomy**—The inguinal canal is  $1\frac{1}{4}$  in long and extends from the internal inguinal ring laterally to the external inguinal ring medially. It lies above the inner half of the inguinal ligament.

The internal or abdominal ring is just above the midpoint of the inguinal ligament. It is an opening in the transversalis fascia and just medial to it is the inferior epigastric artery.

The subcutaneous or external ring lies above and lateral to the pubic crest. It is an opening in the external oblique and through it passes the spermatic cord in the male and the round ligament in the female. They lie lateral to the pubic spine.

The walls of the inguinal canal are—

- 1 Anteriorly external oblique internal oblique in its lateral third
- 2 Posteriorly fascia transversalis in its whole length conjoined tendon or falx inguinalis in its inner two thirds reflex inguinal ligament in its inner third
- 3 The floor inguinal ligament
- 4 The roof arching fibres of the conjoined tendon

The contents of the inguinal canal are the ilio inguinal nerve and the spermatic cord or the round ligament of the uterus. The spermatic cord comprises the internal and external spermatic arteries and the artery to the vas deferens the pampiniform plexus of veins the lymphatic vessels the autonomic nerve fibres the vas deferens.

**INDIRECT INGUINAL HERNIA**—This traverses the inguinal canal and is congenital. The sac is a process of peritoneum surrounded by the coverings of the spermatic cord which are from without inwards—

- a The external spermatic fascia from the external oblique
- b The cremasteric fascia from the internal oblique
- c The internal spermatic fascia from the fascia transversalis

**DIRECT INGUINAL HERNIA**—This leaves the abdominal cavity through the triangle of Hesselbach the boundaries of which are lateral the inferior or deep epigastric artery medial the outer border of the rectus inferior the inguinal ligament.

**NERVE SUPPLY**—The nerve supply of the inguinal region is from the last two dorsal and the first two lumbar nerves via the iliohypogastric the ilio inguinal and the genitocrural.

The last two dorsal nerves run downwards and inwards just above the anterior superior iliac spine between the internal oblique and transversus muscles. They end up by piercing the rectus sheath.

The iliohypogastric and ilio inguinal nerves come from the first lumbar root. They are inferior to the last two dorsal nerves and curve round the body just above the iliac crest gradually piercing the muscles and ending superficially. The ilio inguinal nerve traverses the inguinal canal lying anterior to the spermatic cord.

The genitocrural or genitofemoral nerve comes from the first and second lumbar nerves. It enters the inguinal canal from behind piercing the abdominal ring.

**Field Block for Repair of Inguinal Hernia** *continued***Technique** — Three wheals are made as follows —

- 1 A finger breadth internal to the anterior superior iliac spine
- 2 *Over the spine of the pubis*
- 3 Half an inch above the midpoint of the inguinal ligament

Through wheal 1 a larger needle is introduced vertically backwards until it is felt to pierce the aponeurosis of the external oblique with a slight click. After aspiration 30 ml of solution are injected so that both the ilio inguinal and iliohypogastric nerves are surrounded. At this point a needle introduced perpendicular to the skin will not pierce the peritoneum.

Solution is deposited in all layers including that small area of tissue between the wheal and the anterior superior spine.

Through wheal 2 a larger needle deposits solution in the intradermal and subcutaneous layers in the direction of the umbilicus. This blocks nerve twigs overlapping from the opposite side.

Through wheal 3 a needle is inserted perpendicularly to the skin until it pierces the aponeurosis of the external oblique. At this level 20 ml of solution is injected so that the genito femoral nerve is blocked (Macintosh).

Intradermal and subcutaneous infiltration along the line of the incision may be necessary to get a perfect analgesia.

If the hernia is strangulated or irreducible deeper layers should be injected by the surgeon under vision as he goes along.

The patient may complain of temporary discomfort while the neck of the sac is under tension. This can often be relieved by infiltration of local analgesic solution round the neck.

Hemiorrhaphy can also be performed under paravertebral nerve-block. Injections must be made into the eleventh and twelfth dorsal and the first and second lumbar nerves. If in addition the third lumbar nerve is injected Gallie's operation of fascial suture can be performed.

**FIELD BLOCK FOR REPAIR OF FEMORAL HERNIA**

**Anatomy** — A femoral hernia passes through the femoral canal and the saphenous opening or fossa ovalis, an opening in the deep fascia of the thigh  $1\frac{1}{2}$  in below and  $1\frac{1}{2}$  in lateral to the pubic tubercle.

The femoral canal is the most medial of three compartments the most lateral containing the femoral artery and the intermediate one the femoral vein. The femoral canal is  $\frac{1}{2}$  in long and at its mouth is the femoral ring.

The femoral ring is bounded in front by the inguinal ligament and behind by the pectineus. Laterally by the femoral vein and medially by the lacunar ligament of Gimbernat. Cooper's ligament is a backward extension of the lacunar ligament along the pelvic brim (iliopectineal line) for half an inch.

The ring contains the femoral septum or fatty pad.

The coverings of a femoral hernia are from within outwards the fat from the femoral septum, the prolongation of the fascia transversalis forming the anterior wall of the femoral sheath, the cruriform fascia of the fossa ovalis.

**Technique**—The technique is similar to that for radical cure of inguinal hernia with the addition that the lump in the thigh is surrounded by subcutaneous and intradermal wheals.

Paravertebral block from D 10 to L 3 may also be carried out. It is an excellent procedure for operation on strangulated hernia both inguinal and femoral.

### FIELD BLOCK FOR GASTROSTOMY

Wheals are raised along the costal margin from the xiphisternum to the tip of the 10th or 11th rib—on the left side. From needles inserted through these wheals the tissues between the skin and the peritoneum are infiltrated and subcutaneous infiltration is made by joining the wheals together. Lastly a line of subcutaneous infiltration between the xiphisternum and the umbilicus is made. Solution required is 50–100 ml.

### ILIAC CREST BLOCK

A wheal is raised  $1\frac{1}{2}$  in. from the anterior superior iliac spine on a line joining this spine to the xiphisternum. A needle is inserted laterally first just beneath the skin and then deeper until the ilium is touched. Solution is injected so that it anesthetizes the 12th thoracic iliohypogastric and ilio-inguinal nerves as they lie between the internal oblique and the transversus abdominis muscles.

### FIELD BLOCK FOR APPENDICECTOMY

This is most useful in interval cases but is seldom successful in operation for acute appendicitis.

Two wheals are raised one just above and behind the anterior superior iliac spine the second below the costal margin at the tip of the 10th rib on the right side. The tissues between the skin and peritoneum in this line are infiltrated and subcutaneous infiltration is made between the two wheals and downwards between the lower wheal and the iliac bone. Thus the 10th, 11th and 12th dorsal nerves are blocked together with the ilio-inguinal and iliohypogastric.

Splanchnic block is usually necessary in addition except in the very simplest operations on thin subjects.

### FIELD BLOCK FOR SUPRAPUBIC CYSTOSTOMY

Bilateral rectus sheath block is performed through wheals at the lateral margin of the rectus between the umbilicus and the pubes. The wheals are joined by lines of subcutaneous infiltration. From a wheal just above the symphysis a needle is inserted backwards and downwards keeping its point near the bone. Solution is then injected into the cave of Retzius using 30 ml.

### FIELD BLOCK FOR OPERATIONS ON THE ANAL CANAL

A wheal is made on each side of the anus and 1 in. away from it. From these wheals a subcutaneous rhomboidal zone of infiltration is made. Deep injections are now made from the infiltrated zone in the form of a truncated cone a finger in the rectum preventing perforation of the mucous membrane. The nerve to the external anal sphincter is the perineal branch of the fourth sacral nerve.

Field Block for Repair of Inguinal Hernia *continued*

**Technique**—Three wheals are made as follows —

- 1 A finger breadth internal to the anterior superior iliac spine
- 2 Over the spine of the pubis
- 3 Half an inch above the midpoint of the inguinal ligament

Through wheal 1 a larger needle is introduced vertically backwards until it is felt to pierce the aponeurosis of the external oblique with a slight click. After aspiration 30 ml of solution are injected so that both the ilio inguinal and iliohypogastric nerves are surrounded. At this point a needle introduced perpendicular to the skin will not pierce the peritoneum.

Solution is deposited in all layers including that small area of tissue between the wheal and the anterior superior spine.

Through wheal 2 a larger needle deposits solution in the intradermal and subcutaneous layers in the direction of the umbilicus. This blocks nerve twigs overlapping from the opposite side.

Through wheal 3 a needle is inserted perpendicularly to the skin until it pierces the aponeurosis of the external oblique. At this level 20 ml of solution is injected so that the genito femoral nerve is blocked (Macintosh).

Intradermal and subcutaneous infiltration along the line of the incision may be necessary to get a perfect analgesia.

If the hernia is strangulated or irreducible deeper layers should be injected by the surgeon under vision as he goes along.

The patient may complain of temporary discomfort while the neck of the sac is under tension. This can often be relieved by infiltration of local analgesic solution round the neck.

Herniorrhaphy can also be performed under paravertebral nerve-block. Injections must be made into the eleventh and twelfth dorsal and the first and second lumbar nerves. If in addition the third lumbar nerve is injected Gallie's operation of fascial suture can be performed.

**FIELD BLOCK FOR REPAIR OF FEMORAL HERNIA**

**Anatomy.**—A femoral hernia passes through the femoral canal and the saphenous opening or fossa ovalis an opening in the deep fascia of the thigh  $1\frac{1}{2}$  in below and  $1\frac{1}{2}$  in lateral to the pubic tubercle.

The femoral canal is the most medial of three compartments the most lateral containing the femoral artery and the intermediate one the femoral vein. The femoral canal is  $\frac{1}{2}$  in long and at its mouth is the femoral ring.

The femoral ring is bounded in front by the inguinal ligament and behind by the pectineus laterally by the femoral vein and medially by the lacunar ligament of Gimbernat. Cooper's ligament is a backward extension of the lacunar ligament along the pelvic brim (iliopectineal line) for half an inch.

The ring contains the femoral septum or fatty pad.

The coverings of a femoral hernia are from within outwards the fat from the femoral septum the prolongation of the fascia transversalis forming the anterior wall of the femoral sheath the cribriform fascia of the fossa ovalis.

**Technique**—The technique is similar to that for radical cure of inguinal hernia with the addition that the lump in the thigh is surrounded by subcutaneous and intradermal wheals

Paravertebral block from D 10 to L 3 may also be carried out it is an excellent procedure for operation on strangulated hernia both inguinal and femoral

### FIELD BLOCK FOR GASTROSTOMY

Wheals are raised along the costal margin from the xiphisternum to the tip of the 10th or 11th rib—on the left side From needles inserted through these wheals the tissues between the skin and the peritoneum are infiltrated and subcutaneous infiltration is made by joining the wheals together Lastly a line of subcutaneous infiltration between the xiphisternum and the umbilicus is made Solution required is 50–100 ml

### ILIAC CREST BLOCK

A wheal is raised  $1\frac{1}{2}$  in from the anterior superior iliac spine on a line joining this spine to the xiphisternum A needle is inserted laterally first just beneath the skin and then deeper until the ilium is touched Solution is injected so that it anaesthetizes the 12th thoracic iliohypogastric and ilio-inguinal nerves as they lie between the internal oblique and the transversus abdominis muscles

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### ILIAC CREST BLOCK

A wheal is raised 1½ in from the anterior superior iliac spine on a line joining this spine to the xiphisternum A needle is inserted laterally first just beneath the skin and then deeper until the ilium is touched Solution is injected so that it anæsthetizes the 12th thoracic ilio hypogastric and ilio inguinal nerves as they lie between the internal oblique and the transversus abdominis muscles

### FIELD BLOCK FOR APPENDICECTOMY

This is most useful in interval cases but is seldom successful in operation for acute appendicitis

Two wheals are raised one just above and behind the anterior superior iliac spine the second below the costal margin at the tip of the 10th rib on the right side The tissues between the skin and peritoneum in this line are infiltrated and subcutaneous infiltration is made between the two wheals and downwards between the lower wheal and the iliac bone Thus the 10th 11th and 12th dorsal nerves are blocked together with the ilio inguinal and iliohypogastric

Splanchnic block is usually necessary in addition except in the very simplest operations on thin subjects

### FIELD BLOCK FOR SUPRAPUBIC CYSTOSTOMY

Bilateral rectus sheath block is performed through wheals at the lateral margin of the rectus between the umbilicus and the pubes The wheals are joined by lines of subcutaneous infiltration From a wheal just above the symphysis a needle is inserted backwards and downwards keeping its point near the bone solution is then injected into the cave of Retzius using 30 ml

### FIELD BLOCK FOR OPERATIONS ON THE ANAL CANAL

A wheal is made on each side of the anus and 1 in away from it From these wheals a subcutaneous rhomboidal zone of infiltration is made Deep injections are now made from the infiltrated zone in the form of a truncated cone a finger in the rectum preventing perforation of the mucous membrane The nerve to the external anal sphincter is the perineal branch of the fourth sacral nerve



**FIELD BLOCK FOR CIRCUMCISION**

**Anatomy**—The sensory nerves of the penis are derived from the terminal twigs of the internal pudendal or pudic nerves the dorsal nerves of the penis coming beneath the pubic bone one on each side of the midline lying against the dorsal surface of the corpus cavernosum. The skin at the base of the organ is supplied by the ilio inguinal and perhaps the genitofemoral nerves. In addition the posterior scrotal branches of the perineal nerves run para urethrally to the ventral surface and frænum so four nerves have to be blocked.

**Technique**—An intradermal and subcutaneous ring is injected around the base of the penis the subcutaneous infiltration should precede the intradermal. The dorsal nerve is next blocked on each side by injecting 5 ml of solution into the dorsum of the organ so that the needle point lies against the corpus cavernosum. If the needle pierces the corpus cavernosum pain is experienced. For the ventral injection of the para urethral branches the penis should be pulled upwards and 2 ml of solution injected near the base of the organ into the groove formed by the corpora cavernosa and the corpus spongiosum.

Adrenaline must be used with care and in small amount lest necrosis of tissue results as the arteries of the penis are end arteries.

**FIELD BLOCK FOR TRENDLENBURG OPERATION FOR VARICOSE VEINS**

**Anatomy**—The great saphenous vein begins on the dorsum of the foot ascends just in front of the internal malleolus and joins the femoral vein about 1 in below the inguinal ligament after passing through the fossa ovalis. Near its termination it is joined by the superficial circumflex iliac the superficial external pudendal and the superficial epigastric veins. These are irregular and their number may be added to. The femoral vein is medial to the palpable femoral artery.

The sensory nerve supply of the skin of the front of the thigh is from the first four lumbar nerves via the external femoral cutaneous (L 2 and 3) the genitofemoral (L 1 and 2) the anterior crural or femoral (L 2 3 and 4) the obturator (L 2 3 and 4) and the ilio inguinal (L 1).

**Technique**—Wheals are raised—

- 1 Two finger breadths below and medial to the anterior superior iliac spine
- 2 Two finger breadths external to the pubic tubercle
- 3 Just below the inguinal ligament immediately over the pulsating femoral artery

Wheals 1 and 2 are joined by a line of intradermal and subcutaneous infiltration running just above the inguinal ligament. Similar lines are extended downwards from each wheal near the inner and outer borders of the thigh for 3–4 in.

A needle is inserted perpendicularly through wheal 3 so that it pierces the deep fascia and comes to lie near the femoral artery. The needle is kept moving and 20 ml of \_\_\_\_\_ injected.

into this region so that the anterior crural or femoral nerve is blocked. This may cause some temporary weakness of the quadriceps extensor muscles of the thigh.

If sclerosing solutions are injected into the veins under general anaesthesia, lack of muscular movement may cause pooling of the solution in the deep veins with consequent thrombosis there and sometimes pulmonary embolism. If local analgesia is used, muscular activity quickly sweeps away the sclerosant solution.

**Technique of Block of Iliohypogastric and Ilio-inguinal Nerves —**  
(See ILIAC CREST BLOCK p. 359)

## NERVE BLOCK AT THE UPPER PART OF THE THIGH

(Figs 54-57)

**Anatomy of Lumbar Plexus —**The anterior primary divisions of the lumbar nerves each receive a grey ramus communicans from a lumbar ganglion, while the first and second lumbar nerves are also connected with the trunk by a white ramus.

The anterior primary divisions of first four nerves form the lumbar plexus. A branch from the fourth unites with the fifth nerve to form the lumbosacral trunk, which joins the sacral plexus. It lies in the psoas major, anterior to the transverse processes of the lumbar vertebrae.

The branches of the lumbar plexus are —

**THE ILIOHYPOGASTRIC NERVE** from L 1. It leaves the psoas major, crosses the quadratus lumborum, perforates the transversus abdominis and then divides into lateral and anterior cutaneous branches. Its lateral cutaneous branch supplies the skin on the anterior part of the gluteal region after piercing the internal and external oblique muscles 5 cm. behind the anterior superior iliac spine and just above the iliac crest, while the terminal part of the nerve supplies the skin over the pubic bone after piercing the aponeurosis of the external oblique 2 cm. medial to the anterior superior iliac spine. It does not divide into anterior and posterior branches.

**THE ILIO INGUINAL NERVE** from L 1. This accompanies the iliohypogastric nerve in its early course, lying just inferior to it in close relationship to the iliac crest. About 2 cm. anterior and just below the anterior superior spine it pierces the internal oblique and runs medially behind the aponeurosis of the external oblique. It then passes with the spermatic cord through the inguinal canal and supplies the skin of the upper and medial part of the thigh and the adjacent skin covering the external genitals. It has no lateral cutaneous branch, unlike the iliohypogastric nerve, and in the inguinal canal is sensory.

**THE GENITOCRURAL (GENITOFEMORAL) NERVE** from L 1 and 2. Its genital branch supplies the skin of the scrotum or labium majus and the cremaster muscle. Its crural branch supplies an area of skin on the middle of the anterior surface of the upper part of the thigh.



Fig 54

↑

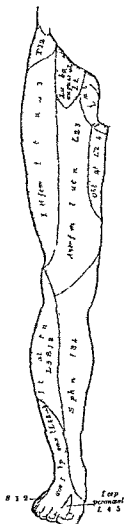


Fig 55

Fig 54—The cutaneous nerves of the right lower extremity. Anterior aspect.

Fig 55—The segmental distribution of the cutaneous nerves of the right lower extremity. Anterior aspect.

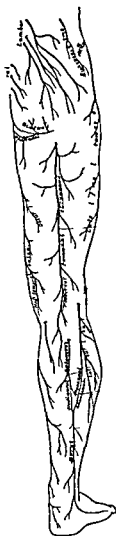


Fig 56

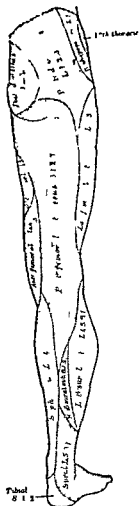


Fig 57

Fig 56—The cutaneous nerves of the right lower extremity. Posterior aspect.

Fig 57—The segmental distribution of the cutaneous nerves of the right lower extremity. Posterior aspect.

(Fig 54-57 from *Clays Anatomy* by kind permission of P. J. or T. B. Johnston)

Nerve block at the Upper Part of the Thigh—Anatomy *continued*

**THE EXTERNAL CUTANEOUS NERVE** of the thigh (lateral femoral cutaneous) from L 2 and 3 (posterior divisions) It supplies the skin of the anterolateral aspect of the thigh as far as the knee anteriorly but laterally not quite so low after passing behind the inguinal ligament and the sartorius muscle just medial and slightly inferior to the anterior superior iliac spine

**THE ANTERIOR CRURAL OR FEMORAL NERVE** from L 2 3 and 4 (posterior divisions) —It emerges from the psoas major passes between it and the iliacus and enters the thigh behind the inguinal ligament and just lateral to the femoral artery from which it is here separated by a slip of the psoas major It has anterior and posterior divisions the latter giving rise to the internal or long saphenous nerve and the medial and intermediate cutaneous nerves of the thigh The anterior crural nerve supplies the skin of the anterior part of the thigh and the anteromedial part of the leg It is motor to the quadriceps femoris

**THE OBTURATOR NERVE** from L 2 3 and 4 (anterior divisions) It supplies an area of skin on the medial aspect of the thigh

**THE ACCESSORY OBTURATOR NERVE** from L 3 and 4 (anterior divisions)

**Anatomy of Sacral Plexus** —The anterior primary divisions of the five sacral and the coccygeal nerves receive each a grey ramus from the sympathetic trunk From the second third and fourth sacral nerves white rami join the pelvic plexuses of the sympathetic the sacral outflow of the autonomic nervous system or the sacral parasympathetics

The sacral and pudendal plexuses are composed of the lumbosacral trunk (L 4 and 5) and the anterior divisions of the five sacral and the coccygeal nerve They lie on the posterior wall of the pelvic cavity between the pyriformis and the pelvic fascia and have in front the ureter internal iliac vessels and the sigmoid colon on the left

The following branches are given off —

Nerve to superior gemellus (L 5 S 1-2)

Nerve to inferior gemellus (L 4-5 S 1)

Nerve to obturator internus (L 5 S 1-2)

Nerve to quadratus femoris (L 4-5 S 1)

Nerve to pyriformis (S 1 and 2)

Superior gluteal nerve (L 4 and 5 S 1)

Inferior gluteal nerve (L 5 S 1 and 2)

Small sciatic nerve (posterior femoral cutaneous) (S 1 2 and 3)

Great sciatic nerve (L 4 and 5 S 1 2 and 3)

Perforating cutaneous nerve (S 2 and 3)

Pudendal or pudic nerve (S 2 3 and 4)

Anococcygeal nerve (S 4 and 5 C 1)

**THE SMALL SCIATIC NERVE** supplies the skin of the lower part of the gluteal region the perineum and the back of the thigh and leg

**THE GREAT SCIATIC NERVE** supplies the skin of the back of the leg and sole of the foot, after dividing into internal popliteal (tibial) and external popliteal (common peroneal) nerves. It leaves the pelvis through the greater sciatic foramen lying between the piriformis and the superior gemellus muscles and occupies the space between the ischial tuberosity and the greater trochanter.

**THE PERFORATING CUTANEOUS NERVE** supplies the skin over the medial and lower parts of the gluteus maximus.

**THE PUDIC (PUDENDAL) NERVE** leaves the pelvis through the greater sciatic foramen, crosses the ischial spine medial to the pudendal vessels and goes through the lesser sciatic foramen. With the pudendal vessels it passes upwards and forwards along the lateral wall of the ischio-rectal fossa in Alcock's canal, a sheath of the obturator fascia. It gives off —

- a The inferior hæmorrhoidal nerve supplying the external anal sphincter and the skin around the anus
- b The perineal nerve supplying the skin of the scrotum or labium majus and small twigs to muscles
- c The dorsal nerve of the penis or clitoris
- d Visceral branches supplying the rectum and bladder. From the second, third and fourth sacral nerves. They communicate with the sympathetic pelvic plexuses. Sometimes called the pelvic splanchnic nerves of Gaskell. Homologous with the white rami communicantes of the thoracic and upper lumbar nerves. The levator ani and the coccygeus and also the external anal sphincter are supplied by the fourth sacral nerve.

**Technique of Femoral Nerve Block** — A wheal is raised a finger breadth to the outer side of the common femoral artery just below Poupart's ligament. A needle seeks for paræsthesia and when found 10 ml of 2 per cent lignocaine is injected. The nerve lies beneath the deep fascia.

Sciatic and femoral nerve blocks in combination are useful in operations on the leg and foot from a point 5 cm below the patella. Operations on or above the knee require in addition external femoral cutaneous and obturator block, the latter being difficult. Manipulations of the lower half of the femur can however be carried out under sciatic and femoral block and the application of a tourniquet to the thigh is painless.

**Technique of Block of External Cutaneous Nerve of Thigh** — A wheal is raised one finger breadth below and medial to the anterior superior spine of the ilium. A needle is inserted perpendicularly to the skin and 1 per cent lignocaine is deposited between the skin and the iliac bone and along the pelvic brim for two finger breadths internally to the anterior superior spine. 10–15 ml of solution is used. The nerve lies deep to the fascia lata of the thigh.

When associated with anterior crural block adequate analgesia is produced for taking skin grafts from the front of the thigh. Alternatively infiltration analgesia of skin of front of thigh can

**Nerve block at the Upper Part of the Thigh** *continued*

be performed using intradermal and subcutaneous injections to cover the desired area

**Technique of Obturator Block**—With the leg slightly abducted a wheal is raised just outside and inferior to the pubic spine and a needle introduced at right angles to the skin until the horizontal ramus of the pubic bone is located. The needle is partly withdrawn and its point directed upwards and outwards and advanced at an angle of 80° to the skin until the upper wall of the obturator canal is felt. The needle is passed for  $\frac{1}{2}$  in through the canal and 15 ml of 2 per cent lignocaine is injected with the needle gently moving. Paræsthesia is unusual. This block is difficult and it is probably preferable to paralyse it by performing a para-vertebral somatic block of the second third and fourth lumbar nerves. Successful block is shown by weakness when the patient attempts to adduct the leg.

Block of the above four nerves will produce analgesia of the lower extremity below the level of the symphysis pubis.

For insertion of a Smith Petersen pin the line of incision is infiltrated down to the bone with 0.5 per cent lignocaine or 1:1000 amethocaine. Solution is injected between the ends of the fractured neck from above the great trochanter and from a point just external to the pulsating femoral artery.

**Technique of Sciatic (Great and Small) Block**—Patient lies on sound side with hip slightly flexed. A line is traced between the upper extremity of the great trochanter and the posterior superior iliac spine. From the midpoint of this line a perpendicular is dropped 3 cm long and at its end a wheal is raised and a needle introduced at right angles to the skin plane until it reaches the ischial spine 2 to 3 in from the skin surface. The nerve lies on this area of bone so that paræsthesia must be elicited before the needle strikes bone. 10–15 ml of 2 per cent lignocaine is then injected.

A second surface marking is the junction of the medial third with the lateral two thirds of a line joining the ischial tuberosity to the greater femoral trochanter—the needle being inserted at right angles to the skin surface until paræsthesia are felt by the patient.

The block is useful for reduction of fractures round the ankle.

It also causes almost complete vasoconstrictor paralysis of the foot and is better safer and less painful than lumbar sympathetic block for this purpose. A rise in skin temperature starts in ten minutes and is maximal in 20–30 minutes. Sciatic nerve block gives analgesia of the whole foot with the exception of an area of skin over the internal malleolus supplied by the internal saphenous branch of the femoral nerve.

**Technique of Perineal Block**—The perineal branch of the pudic nerve supplies the skin of the anterior part of the perineum in front of the anus. With the patient in the lithotomy position a wheal is raised on each side just internal to the ischial tuberosity. Through it a needle is advanced about 1 in and 10–15 ml of 2 per cent lignocaine is injected on each side.

The ilio-inguinal nerve can be blocked as it curves over the tendon of the adductor longus close to the pubic bone the nerve supplies the skin over the symphysis or its immediate surroundings

### ANKLE BLOCK

A subcutaneous and intradermal wheal is raised circumferentially around the ankle just above the internal malleolus

ANTERIOR TIBIAL NERVE is blocked by inserting a needle midway between the most prominent points of the internal and external malleoli on the circular line of infiltration It is directed medially towards the anterior border of the internal malleolus and solution is injected between the bone and the skin par æsthesia should be elicited if possible

The amount of solution used should be 10-15 ml

POSTERIOR TIBIAL NERVE is blocked by 10 ml of solution introduced through a point on the circular wheal just internal to the tendo Achillis The needle is inserted forwards slightly outwards towards the posterior aspect of the tibia near which the solution is deposited

After waiting ten minutes ankle block is suitable for operation on the foot.

Posterior tibial block is useful for testing vasodilatation of the foot.

Following infiltration of any peripheral nerve there results a vasomotor paralysis over the area of anæsthetized skin

### BLOCK FOR HALLUX VALGUS

A wheal is raised near the proximal end of the first intermetatarsal space superior surface and from this solution is deposited between the two layers of skin dorsal and plantar as far forwards as the web between the toes More solution is injected between the dorsal skin and the first metatarsal bone A second wheal medial to the first one on the internal aspect of the metatarsal is raised and injection made between it and the bone and between the plantar skin and the bone A 10-15 minute pause is made before the operation is commenced From 30 to 50 ml of solution is used

### LOCAL ANALGESIA FOR REDUCTION OF CLOSED FRACTURES

After localizing the exact site of fracture by means of X rays a wheal is raised near the fracture and a needle introduced into the hæmatoma between the broken bone ends Aspiration of old blood confirms the position and must be obtained injection is then made of 1.5 per cent lignocaine or 2 per cent procaine For Colles's fracture the amount required is 20 ml injection should be made from the extensor aspect of the wrist and in addition a few ml should infiltrate the ulnar styloid for Pott's fracture 40 ml of solution with hyaluronidase can be used for fractured femur 50 ml Cases of recent fracture are the most suitable for this method of reduction Adrenaline 1-100 000 should be added to analgesic solution 1-2 per cent lignocaine works well Good results are claimed for the addition of 1000 units of hyaluronidase to each 20 ml of solution With procaine analgesia lasts two to three hours and comes on after ten minutes



## TOPICAL ANALGESIA FOR BRONCHOSCOPY AND ŒSOPHAGOSCOPY

*Cocaine Sensitivity Test*—Inject 10 mg hypodermically e.g. 0.25 ml of 4 per cent solution. For amethocaine sensitivity inject 2 mg e.g. 0.2 ml of 1 per cent solution. If pulse rate rises markedly patient is presumably sensitive to the drug.

After nembutal and atropine or other similar premedication the patient is given a tablet of amethocaine (65 mg—1 gr) to suck. The gums, tongue, palate and pharynx are now sprayed with 4 per cent xylocaine, 4 per cent cocaine, 1 per cent amethocaine, 2 per cent butyn or some other suitable analgesic solution. He can be given Xylocaine Viscous to swallow, a 2 per cent solution in a mucilage base, very pleasantly flavoured.

Cocaine should be used carefully in a maximum dosage of 20 mg per stone (3 mg per kilo). Amethocaine in 1 per cent solution should not exceed 8 ml and should have adrenaline 1:1000 added to it in proportion of 4 ml amethocaine to 1 ml of adrenaline. These topical solutions should not be used on inflamed or traumatized surfaces. Xylocaine in 4 per cent solution gives good results and is not very toxic.

After a short interval the pyriform fossæ are made analgesic by application of swabs soaked in the solution (with excess of it removed) applied on a curved applicator or with Krause's laryngeal forceps. After this has been done and after spraying the epiglottis and the cords a swab is gently introduced into the glottis and held there for a short while so that its analgesic solution completes the block of the superior laryngeal nerve endings.

In addition if bronchoscopy is to be performed the trachea must be anesthetized by

1. The transtracheal method of Canuyl (1920) which consists in inserting a fine hypodermic needle through the middle line of the neck into the trachea. The needle should be  $1\frac{1}{2}$  in long so that if breakage occurs which is usually near the hub the remains of the shaft can be easily removed. Aspiration of air proves its presence in the trachea. An injection of 2–3 ml of 4 per cent cocaine is now made after a deep expiration and the patient told not to cough until the solution has trickled down the trachea into the bronchi. Complications are (1) Infection (2) A broken needle (3) Surgical emphysema.
2. By instillation from a laryngeal syringe using an indirect laryngeal mirror.
3. By passing a No. 1 endotracheal tube from the cocaineized nose into the trachea and instilling down it via a length of fine polythene tubing a few ml of solution near the carina.

For œsophagoscopy and gastroscopy the intratracheal injection is omitted and the patient is given 2 ml of 4 per cent cocaine to swallow instead. Should toxic signs develop owing to the drug oxygen must be rapidly introduced into the alveoli and a small dose of intravenous barbiturate injected.

Regional analgesia is probably safer than general anaesthesia for these examinations as a conscious cough reflex is maintained so that the air passages can be kept free from blood etc. It enables the movements of the vocal cords to be seen also.

As an aid to comfortable endoscopy under local analgesia Churchill Davidson\* advises the concurrent use of divided doses of pethidine (30-100 mg) given intravenously. Addition of hyaluronidase (10 turbidity reducing units per ml local analgesic solution) reduces the amount of analgesic drug required and speeds the onset of analgesia †

In children who may be susceptible to cocaine and its substitutes such examinations are better made under avertin or rectal paraldehyde followed if necessary by gas oxygen a volatile anaesthetic and a muscle relaxant. Ether air followed by trilene air while the bronchoscope is in the air passages is probably as safe a technique as any the slight theoretical risk of explosion notwithstanding.

Thiopentone must only be used in such examinations if topical analgesia is efficiently done beforehand and relaxants used in addition lest dangerous laryngeal spasm should occur. After topical laryngeal analgesia instruction must be given to the patient not to eat or drink for at least three hours i.e. until the sentinel of the larynx has returned to duty.

Mushin has used bilateral vagus block for these examinations.

Even without the administration of extra oxygen oxygen saturation of the blood increases during bronchoscopy performed under local analgesia ‡

### TOPICAL ANALGESIA OF THE URETHRA

Barbiturate premedication helps to prevent symptoms of intoxication due to local analgesic drugs (Tatum and others 1925). Cocaine can be used in the absence of recent instrumentation or other types of existing or potential trauma where these exist 2 per cent metylocaine is safer. Reactions from cocaine in this region are not uncommon.

**Technique**—30 ml of a mixture of 0.5 per cent cocaine and 0.5 per cent sodium bicarbonate or 2 per cent metylocaine is instilled into the urethra with a glass urethral syringe. Sufficient volume is injected to distend the anterior urethra and allow some to be massaged back to the posterior urethra.

The syringe is removed and a penile clamp applied to keep the solution in contact with the urethral mucosa. This is removed after five minutes and an applicator with cotton wool soaked in 10 per cent cocaine is placed inside the meatus and allowed to remain for a further five minutes.

A useful preparation is Xylocaine Gel a 2 per cent preparation put up in 30 ml tubes. The plastic nozzle supplied by the makers of the preparation is boiled and by its aid the paste is squeezed into the urethra. A clamp is applied to the penis and the paste gently massaged into the posterior urethra. After an interval

Churchill Davidson, H. C. *Anaesthesia* 1952 7 237  
 † Howla, A. W. S., and Papper, E. M. *Anesthesiology* 1951 12 688  
 ‡ de Kornfeld, T. J. and Siebecker, A. L. *Ibid* 1957 18 466

**Topical Analgesia of the Urethra—Technique continued**

of 10–15 minutes instrumentation can begin. It is sometimes helpful to pass a small catheter into the urethra connected to the plastic nozzle so as to deposit paste in the posterior urethra. Each tube contains 300 mg. of the drug.

Nupercaine 0.5 per cent in fragacanth paste can be used instead a volume of 15 ml. being injected and held by a clamp. For patients who react abnormally to ordinary local analgesics 2 per cent solution of pyribenzamine the antihistamine give reasonable analgesia without toxic reactions other than occasional drowsiness.

**EXTRADURAL SACRAL BLOCK (CAUDAL BLOCK)**

This method of analgesia was introduced by Cathelin and Sicard in 1901 and was used by Schlumpert of Freiburg in 1913 and in obstetrics by Stoeckel in 1909 and by Lawen. Has been used in animals especially in cattle since 1925.

**Anatomy of Sacrum**—A large triangular bone formed by the fusion of the five sacral vertebrae articulating above with the fifth lumbar vertebra and below with the coccyx.

Pelvic surface is concave from above downwards and from side to side. It is crossed by four transverse ridges at the lateral extremities of each being an anterior sacral foramen directed forwards and laterally. Each foramen opens into the sacral canal and transmits an anterior division of a sacral nerve.

Posterior surface is convex and down its middle line runs the median sacral crest with its three or four rudimentary spinous processes. The laminae of the fifth and sometimes of the fourth sacral vertebrae fail to fuse in the midline the deficiency thus formed is known as the sacral hiatus. The tubercles representing the inferior articular processes of the fifth sacral vertebra are prolonged downwards as the sacral cornua. These cornua with the rudimentary spine of the fourth vertebra above bound the sacral hiatus. Four posterior sacral foramina correspond with the anterior foramina. Each transmits a posterior sacral nerve division and communicates with the sacral canal.

Lateral surfaces are broader above than below and are ear shaped. Each articulates with the ilium.

Base of the sacrum is directed upwards and forwards and articulates with the fifth lumbar vertebra. The central portion forms the sacral promontory this being flanked by the sacral ala on each side. The ala represents the transverse and costal processes of the first sacral vertebra.

Apex is directed downwards and articulates with the coccyx.

Coccyx represents four rudimentary vertebrae—sometimes three or five.

Female sacrum is shorter and wider than the male sacrum. It is directed more obliquely backward thus increasing the size of the cavity of the pelvis.

**SACRAL CANAL** is a prismatic cavity running throughout the length of the bone and following its curves. y it is

triangular on section and is continuous with the lumbar vertebral canal. Its lower extremity is the sacral hiatus closed by the posterior sacrococcygeal membrane. Fibrous strands some times occur in the canal and divide the extradural space into compartments. These may account for some cases of failure to produce uniform analgesia. Its anterior wall is formed by fusion of the bodies of the sacral vertebrae. Its posterior wall by fusion of the laminae. The hiatus results from lack of fusion of the laminae of the fifth sacral vertebra. On each lateral wall of the canal four foramina are present which divide in the form of a V into anterior and posterior sacral foramina. The contents of the sacral canal are —

- 1 The dural sac which ends at the lower border of the second sacral vertebra on a line joining the posterior superior iliac spines. The pia mater is continued as the filum terminale
- 2 The sacral nerves and the coccygeal nerve with their dorsal root ganglia
- 3 A venous plexus formed by the lower end of the internal vertebral plexus. These vessels are more numerous anteriorly than posteriorly and so needle point should be kept as far posteriorly as possible
- 4 Areolar and fatty tissue—more dense in males than females

Each sacral nerve is provided with a thick sheath from the dura. The sacral hiatus is a triangular opening with apex upwards formed by the fourth sacral spine and a sacral cornu on each side below and laterally. It is covered over by the sacrococcygeal ligament which is pierced by the coccygeal and fifth sacral nerves. This is superior to the sacrococcygeal junction usually about  $1\frac{1}{2}$ –2 in from the tip of the coccyx and directly beneath the upper limit of the intergluteal cleft. Anatomical abnormalities of the sacrum are not uncommon. They include (1) Upward displacement of the hiatus (2) Pronounced narrowing of the sacral canal making needle insertion difficult (3) Ossification of the sacrococcygeal membrane (4) Absence of the bony posterior wall of the sacral canal due to failure of fusion of laminae

The average capacity of the sacral canal is 34 ml in males and 32 ml in females. Its average length is 3 to 4 in.

When a local analgesic solution is injected into the sacral canal it ascends upwards in the extradural space for a distance proportional to the volume of solution the force of injection the amount of leakage through the intervertebral foramen and the consistency of the connective tissue in the space. While the first two are controllable the last are not so precise placement of the solution is impossible and sometimes leads to unexpected results. As the extradural space communicates through the intervertebral foramina with the paravertebral spaces (which are not themselves intercommunicating) extradural block affects

- (1) Anterior nerve roots
- (2) Posterior nerve roots and

**Extradural Sacral Block—Anatomy of Sacrum** *continued*

root ganglia (3) The mixed spinal nerves (4) The ganglia of the sympathetic chain (5) The grey and white rami communicantes (6) The visceral afferent fibres accompanying the sympathetic fibres

**Technique of Injection**—The patient is in the prone position with hips slightly flexed over two pillows. To prevent tensing of the gluteal muscles the patient should be asked to abduct his legs and turn his toes in. Other positions are the lateral knee-chest and knee elbow. After cleaning and towelling the tip of the coccyx is identified and the triangular sacral hiatus palpated about  $1\frac{1}{2}$  to 2 in above it. The hiatus must be clearly visualized. Its anatomy is not constant: sometimes it is larger, sometimes smaller than normal. It is difficult to feel in fat patients. If necessary a dose of thiopentone can be injected into a Mitchell needle just before the block is commenced.

A wheal is raised over the hiatus using no more than 2 min of solution as oedema obscures the landmarks. A needle such as a Number 1 is inserted through the sacrococcygeal membrane so that it makes an angle of about 20° with the line drawn at right angles to the skin surface. Once through the membrane the needle is depressed a further 45° towards the intergluteal cleft and the needle is inserted into the sacral canal keeping in the midline. The point must not ascend higher than the line joining the posterior superior iliac spines lest the dura which ends at this level be entered. Occasionally the dural sac extends lower down than the level of the second piece of the sacrum. The mean distance between the apex of the hiatus and the dural sac is 4.5 cm. In many cases the needle used for the skin wheal can be used to enter the sacral canal.

After aspiration tests for blood and cerebrospinal fluid have been proved negative a test dose of 8 ml of solution is injected. Should blood flow through the needle its position must be slightly altered. Should cerebrospinal fluid appear the method of analgesia must be abandoned and subarachnoid injection substituted for it. In suitable cases the proper amount of drug being introduced into the theca through the sacral needle.

Five minutes after the test injection movement of the toes is called for. If this is present a subarachnoid block has not resulted and the needle point is not in the theca: further injection can then be proceeded with. When the needle is correctly placed injection is easy: no great force being required to depress the plunger of the syringe. Should the needle be posterior to the canal a tumour is raised over the sacrum as the injection proceeds. Injection of a few ml of air will produce surgical emphysema with its crepitus: if the needle is superficial to the canal. If the needle point comes to lie between periosteum and bone the force needed for injection will be great—a sure sign of an incorrect position. Young males because of the tough nature of their peridural fat require larger doses injected with greater force than females.

**Drugs** —Metycaine in 1½ per cent solution in normal saline or Ringer's solution is very satisfactory. Procaine 2 per cent in normal saline can be used while if a prolonged effect is wanted nupercaine 1-500 in normal saline or 0.15 per cent amethocaine hydrochloride in saline can be employed with a maximal dosage of 50-60 ml. Adrenaline should be added. Lignocaine 1 to 1.5 per cent solution is excellent giving a rapid onset and a profound degree of analgesia. Its effects can be prolonged if amethocaine powder is added to make a strength of 0.1 per cent or 0.2 per cent. Intracaine 5 per cent in corn oil has been recommended for rectal surgery the dose being 20-30 ml to which is added 0.5-1 ml of 1-500 adrenaline in oil. About fifteen minutes are taken to give the injection of the warmed oil and analgesia lasts for 6-8 hours with hypalgesia going on for a further 10-12 hours.\*

**DOSAGE** —Level of analgesia is governed by —

- 1 Quantity of solution
- 2 Speed of injection
- 3 Gravity

Poor risk cases require a smaller dose by at least 20 per cent

**Low Block** 1.e. up to L 2 to L 4 for operations on anus rectum perineum or urethra circumcision vaginalplastics etc inject 30 ml of 1 per cent or 1.5 per cent lignocaine slowly. In 10 minutes analgesia will develop and will last one hour. 20 ml of solution is sufficient for the average case of piles or anal fissure.

**Mid Block** 1.e. up to T 10 for operation on lower limbs pelvic organs hernia etc inject 30 ml with table in slight Trendelenburg tilt. In 5 minutes inject a further 10-20 ml fairly rapidly. For operations on lower extremities the tilt can be reversed.

**High Block** 1.e. up to T 4-5 for operations on upper abdomen inject 30 ml and pause 5 minutes. Then tilt the table and inject a further 30 ml. After a second similar pause inject a third dose of 10 ml. High block may take half an hour to develop and should last a further 60-90 minutes. Motor block is not marked but as the reflex arc is broken no reflex rigidity of the muscles of the anterior abdominal wall results from stimuli. The rami communicantes are paralysed and the blood pressure falls though not so markedly as after subarachnoid block. A pressor drug should be at hand to be used in suitable cases. Respiratory paralysis is not a primary occurrence.

Toxic reactions to the drugs used are sometimes seen as dosage is fairly large. An occasional twitch requires no treatment but convulsions should be combated with intravenous hexobarbitone or thiopentone. Collapse due to cardiovascular depression consequent on reduction of the blood pressure is treated on the usual lines by elevating the lower limbs and injecting a pressor drug.

## Dural Sacral Block—Drugs continued

The extent of analgesia is not by any means related only to the volume of solution injected but also to the volume which leaks through the sacral foramina and the intervertebral foramina and to the degree of the lumbo sacral angle.\* The best control of the height of analgesia is obtained by the use of the continuous technique

### Advantages —

- 1 Absence of post operative headaches
  - 2 Less cardiovascular depression than with subarachnoid analgesia
- The method is excellent for cystoscopies hæmorrhoidectomies and gynæcological plastic operations and is most highly recommended by the present author
- It is also most useful for forceps deliveries in obstetrics owing to the excellent relaxation of the cervix and pelvic floor and perineum
- In children test dose varies according to age and size from 2 ml to 4 ml Supplementary dose varies from 8 ml to 15 ml It has been successfully used to lower the blood pressure in children with acute nephritis Such dosage should lead to paralysis of vasoconstrictors of legs and some of the white rami supplying the suprarenal glands—up to T9
- It can be used for orthopædic operations on the foot for ligations of varices and for the prolonged relief of post operative pain after e.g. hæmorrhoidectomy in this case a plastic catheter is left in the sacral canal and through it doses of 12 ml of local analgesic solution can be injected as required
- Its therapeutic indications include low backache sciatica vaso spastic disease of the lower limbs white leg acute anuria sometimes and intractable pelvic pain using up to 60 ml of proctocaine †

### Disadvantages —

- 1 Length of time taken for development of analgesia
- 2 Lack of accurate control of height of analgesia
- 3 Muscular relaxation not maximal in mid and high blocks although it is excellent in low blocks
- 4 Technical difficulty
- 5 Risk of inadvertent subarachnoid injection
- 6 Hypotension The method should not be used in patients with a weak myocardium
- 7 It produces complete flaccidity of the anal sphincters a condition unpopular with some surgeons doing such operations as cure of fistula in ano

## CONTINUOUS CAUDAL (EXTRADURAL SACRAL) BLOCK

This is chiefly used to produce painless labour Single extradural sacral injections have been used for many years in obstetrics (Stoeckel 1909 Schlumpert 1913) but the continuous technique was introduced in 1942 by Hingson and Edwards

**Autonomic Nerve supply of Uterus —**

- 1 Visceral efferent sympathetic (motor to upper uterine segment)  
The middle thoracic segments from T 3 downwards perhaps as low as L 2 via splanchnic nerves and coeliac aortic renal and hypogastric plexuses and thence with blood vessels to the great cervical ganglion of Frankenhauser. In addition some of the motor supply of the body of the uterus may come from below the sensory supply i.e. T 11 L 2. The inferior hypogastric plexus pelvic plexus or plexus of Frankenhauser is situated on each side of the rectum in the base of the broad ligament. The cervix and lateral vaginal fornix are medial relations. Each plexus is a thin sheet. The parasympathetic roots are the pelvic splanchnic nerves while the preganglionic sympathetic roots come from cells in the last three thoracic and first two lumbar segments of the cord.
- 2 Visceral afferent sympathetic (sensory to uterus) Block of these eases pain of the first stage of labour with exception of those near the end of this stage. Eleventh and twelfth thoracic (and possibly first lumbar). Fibres go from uterus (ganglion of Frankenhauser) via sympathetic nerves to pelvic hypogastric and aortic plexuses enter the sympathetic chain at the level of L 5 and ascend in the chain entering the cord via the white rami of T 11 and T 12 and the eleventh twelfth posterior thoracic roots and thence up posterior columns of cord (J. G. P. Cleland 1933). The fibres then cross over to the spinothalamic tract on the opposite side of the cord and ascend to the thalamus and thence to the frontal and parietal areas of the cortex. Block of the sympathetic chain between L 5 and T 12 gives the same freedom from first stage labour pains as block of the eleventh-twelfth thoracic ganglia.
- 3 Visceral afferent and efferent parasympathetic (inhibitory to uterus sensory and motor to cervix sensory to upper birth canal). Second third and fourth sacral nerves directly to great cervical ganglion of Frankenhauser.

**Somatic Afferent Nerves of Lower Birth Canal.—**

The inferior hæmorrhoidal perineal and dorsal nerve of the clitoris—from the pudendal nerve (S 2 3 4)

The ilio inguinal (L 1) and the genitocrural (L 1 2)

There are twenty nerves transmitting labour pains viz—(a) The visceral afferents of the eleventh and twelfth thoracic—pain of uterine contraction. (b) The posterior roots of the second third and fourth sacral nerves carrying pelvic afferents (H<sub>3</sub> Head)—pain of cervical dilatation. (c) The hæmorrhoidal perineal and pudendal branches of the sacral plexus—pain of perineal stretching. (d) The ilio inguinal and genitocrural branches of the lumbar plexus.

The method should be commenced (1) When presenting part is engaged. (2) When cervix is dilated to the size of half a crown (in primiparæ). (3) When contractions are occurring regularly at not more than five minute intervals and are of half minute duration.



## REGIONAL ANÆSTHESIA

### Dural Sacral Block—Drugs continued

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**Continuous Caudal Block** *continued*

**Technique** —With patient in the lateral knee-elbow or knee-chest position a malleable spinal needle is inserted into the sacral canal for 1-2 in. To this is attached a closed system comprising a length of fine bore thick walled rubber tubing a syringe and a reservoir containing the analgesic solution. The needle is taped between the buttocks and the patient turned on to her back. Injections are not begun until labour is well established.

**Solutions used** —

- 1 Metycaine 1½ per cent in normal saline or Ringer's solution
- 2 Procaine 2 per cent in normal saline
- 3 Lignocaine 1 to 1.5 per cent 20 ml with adrenaline 1-100 000 to 1-200 000. This gives a block up to T11 in most cases patients in labour showing a higher level of analgesia than normal women. Repeated injections have a decreasing effect.

If 0.15 per cent amethocaine is used probably one or two separate injections will carry the patient through the latter part of her labour avoiding the need for the continuous technique.

Test dose 8 ml of solution i.e. metycaine 120 mg or procaine 160 mg

Initial dose 30 ml injected in 60 seconds if test dose shows after 10 minutes no evidence of subarachnoid injection having been given. This usually produces freedom from pain in 10 to 15 minutes.

To maintain this subsequent doses of 20 ml are given on an average every 30-40 minutes.

Labour is retarded if analgesia rises higher than half way between the pubis and the umbilicus i.e. higher than or including the tenth dorsal segment as it will affect motor fibres supplying the upper uterine segment. Unilateral analgesia may occur.

The following modifications have been suggested —

- 1 Introduction of a ureteric catheter or plastic tube into the sacral canal through a wide bore needle which is afterwards withdrawn over the catheter (Block and Rochberg 1943; Manalan 1942 and later Adams, Lundy and Seldon 1943). This is less traumatic than an indwelling needle and less likely to break or slip into the theca.
- 2 Maintenance of analgesia by a continuous drip instead of repeated injections. A rate of 8-12 per minute is average. Speeding of drip suggests slipping of needle into theca.
- 3 Addition of adrenaline to solution to prolong its effect. Some workers avoid adrenaline because of its relaxing effect on the uterus.
- 4 Addition of nembutal in early labour to minimize toxic effect of analgesic drugs and for its sedative effect.

For Caesarean section 1 per cent metycaine is substituted for 1½ per cent solution or 1 per cent xylocaine is used. After test dose of 10 ml a primary dose of 40 ml is injected followed by 30 ml every ten minutes until analgesia reaches to the costal margins. The operation must not be begun until a proper height of analgesia has been obtained. The method demands freedom from rush and absence of adherence to a time schedule.

drip should be routine and ephedrine 25 mg can be used a larger dose may inhibit tone of uterus Oxygen inhalation (100 per cent) should be given before and during the operation

**Results**—In ideal cases labour is not retarded the perineum is relaxed from the outset the first and third stages are shortened although the second stage is prolonged frequently requiring forceps delivery—which is rendered easy by the relaxation of the pelvic floor and perineum analgesia being adequate for the forceps extraction and episiotomy respiration of the foetus is spontaneous at birth there is some fall in blood pressure there is complete freedom from pain throughout labour Hingson claims that in 90 per cent of cases pain at the end of the first stage second stage pain and pain from repair of perineal tears can be abolished completely It should not be used for early pain of the first stage

#### **Limitations and Dangers of Continuous Caudal Analgesia —**

- 1 Foetal hypoxia due to hypotension of mother Maternal blood pressure must not be allowed to fall below 80 mm Hg systolic fall in blood pressure must be treated by pressor drug posture elevation of the legs and intravenous fluids The signs of hypoxia in the foetus (a) Bradycardia (b) Increased movements (c) Increased meconium
  - 2 Broken needles or plastic tubing
  - 3 Arrested or prolonged labour due to too high spread of solution or its leakage through the anterior sacral foramina on to the ganglion of Frankenhauser
  - 4 Increased incidence of occipito posterior positions
  - 5 Increased incidence of operative deliveries
  - 6 Massive spinal analgesia If this occurs patient becomes pale and blue and later apnoeic The chief danger is anoxia circulatory collapse being some minutes away Routine resuscitation is as follows —
    - a Direct vision intubation—larynx is insensitive
    - b Rhythmical inflation with oxygen
    - c Give blood pressure-raising drug
    - d Turn patient on to side and withdraw 20–40 ml of cerebro spinal fluid
    - e Give intravenous drip
  - 7 Infection
  - 8 Drug susceptibility on part of the mother and also of the foetus from transplacental contamination
  - 9 Maternal hypotension and circulatory depression Elevation of the legs to a right angle results in an efficient autotransfusion
- In spite of these dangers the stillbirth and neonatal death rates are quoted as being less in cases delivered under continuous caudal analgesia than in cases delivered under other forms of analgesia and anaesthesia or under none at all

#### **Special Indications —**

- 1 In patients with cardiac disease
- 2 In patients with nephritis

**Continuous Caudal Block—Special Indications** *continued*

- 3 In patients with toxæmia It lessens hypertension and water retention and is sufficient for painless delivery either per via naturales or by Cæsarean section
- 4 In patients with pulmonary disease
- 5 In breech extractions
- 6 For premature labour
- 7 In cases of rigid slowly dilating cervix and uterine inertia
- 8 For forceps delivery—by one shot rather than continuous technique
- 9 In emergency Cæsarean section when the patient has a full stomach

**Contra indications —**

- 1 Patients who have quick easy labours
- 2 Unstable neurotic or frightened patients who will not co-operate—e.g. in second stage patients must be told when to bear down and must help themselves in this respect
- 3 Deformity of sacral region—it is impossible to enter the sacral canal of some patients In expert hands failures occur in about 10 per cent of cases
- 4 Disease of the central nervous system
- 5 Obesity—because of difficulty in inserting needle
- 6 Local infection
- 7 Gross anæmia
- 8 Placenta prævia and accidental hæmorrhage because of the relaxation the method produces in the lower uterine segment
- 9 Severe disproportion
- 10 When intra uterine manipulations are anticipated—because of the increase in uterine tone
- 11 Susceptibility to local analgesic drugs

During the labour a close watch must be kept on the foetal heart and the maternal condition especially the blood pressure The technique must only be employed in well equipped hospitals and by experienced anæsthetists and obstetricians Facilities for the treatment of collapse—oxygen intravenous fluids etc—must be at hand

**Uses in Therapeutics of Caudal and Continuous Caudal Block (Hingson\*) —**

- 1 To control eclampsia It reduces the blood pressure stops convulsions relieves blurred vision and headaches diminishes pulmonary œdema relieves failing heart increases renal output and abolishes labour pains
- 2 To relieve oliguria and anuria associated with reflex renal ischæmia
- 3 In post-operative paralytic ileus and distension
- 4 In arterial embolism of lower limbs
- 5 In thrombophlebitis of lower limbs
- 6 To aid prognosis of sympathectomy in essential hypertension
- 7 For prolonged relief of post-operative or post traumatic pain
- 8 For facilitating removal of rectal faecal impaction
- 9 For treatment of renal colic by block up to T 8 and forcing fluids

Galley\* suggests that caudal block gives good results in cases of chronic cold feet phlegmasia alba dolens vasospastic disease of the legs and diabetic neuropathy. Although the effects of single injections were transitory the relief of symptoms in his cases frequently lasted a considerable time owing to the interruption by the block of a self perpetuating vicious circle. The drug used is usually procaine and the amount about 50 ml which should be warmed before injection into the sacral canal. The types B and C fibres are more susceptible to blocking than the A type fibres.

Other sympatholytic drugs used to remove vasomotor tone from peripheral vessels are —

**PRISCOL (Priscoline)** (2 benzyl 4, 5 amidazoline hydrochloride) — An efficient peripheral vasodilator acting on skin vessels even when they are denervated. Has been successfully used in cases of acute arterial occlusion and has been given (by mouth if necessary) both before and after sympathectomy.

**DIBENZYL LINE** (*N* phenoxyisopropyl *N* benzyl betachlorethyl amine hydrochloride) — Formerly known as 688A it is related to dibenamine and is a powerful sympatholytic and adrenolytic drug which can be given intravenously and by mouth.

M Kenny has recommended the injection of up to 50 ml of procaine into the sacral canal of patients who complain bitterly of intolerable pain due to malignant disease of the pelvis. The bladder and anal sphincters are unlikely to be permanently paralysed by the technique. Pain relief is not always perfect.

### TRANS SACRAL BLOCK

First performed by Pauchet and Lâwen in 1909 this involves blocking the sacral nerves through the posterior sacral foramina. It is usually associated with extradural sacral (caudal) block but is frequently quite unnecessary. Solution deposited in the sacral canal through the sacral hiatus usually producing excellent analgesia. It is however useful when an extradural sacral block is required but cannot be induced because of the difficulty of introducing a needle into the sacral canal.

**Technique** — The posterior superior iliac spines are located as the patient lies prone with his pelvis supported on a sandbag. In the obese a dimple overlies the spine. The second foramen is a finger breadth caudad and a finger breadth medial to the spine. The third, fourth and fifth foramina are one finger breadth apart on the same line while the first foramen is a finger breadth above the spine and a similar distance medial to it. The fifth foramen is usually between the sacrum and the coccyx.

The foramina underlie a line a finger breadth lateral to the median line posteriorly.

To prevent pain caudal block should be induced a quarter of an hour before the needles for the trans-sacral block are inserted.

In intractable pain and in severe sciatica a watery solution of phenol has been injected with success into the first and second sacral foramina.

**Trans sacral Block—Technique continued**

Wheals are raised and needles introduced no more than half way through each foramen. With a negative aspiration test for cerebrospinal fluid and blood the following amounts of 0.5 per cent xylocaine, 1 per cent procaine or metycaine are injected on each side —

First sacral foramen	15 ml	Second foramen	10 ml
Third foramen	4 ml	Fourth foramen	3 ml
Fifth foramen		2 ml	

Lundy recommends the injection into the sacral canal of 30 ml of 1 per cent procaine followed by trans-sacral injections into the fourth, third and second foramina (starting from below because of the earlier analgesia low down produced by the caudal injection). His opinion is that if the solution used is 1 per cent procaine toxic effects are unlikely and should they occur e.g. convulsions are not serious.

Block of the posterior divisions of the sacral nerves by long acting agents by the trans sacral route is said to be useful in the treatment of the hypertonic bladder in paraplegics. Block of S3 or S2 and S3 on each side will enable a patient to micturate.

**Analgesia of the Peritoneal Cavity by Lavage**—Amethocaine 0.1 per cent solution, 0.5 per cent lignocaine, 0.5 per cent to 1 per cent procaine are used. Volume 200 ml. This is poured into the peritoneal cavity and the peritoneal edges are drawn together. After 5 to 8 minutes the solution is sucked out. Results: relaxation of the peritoneum, contraction of the bowel, absence of reflex response from visceral trauma. Toxic reactions are said to be rare. Good results are not seen in cases of peritonitis or where the bowel is grossly distended.

**INTRAVENOUS LOCAL ANÆSTHESIA**

Introduced by Bier in 1909. Involves the injection of a local anæsthetic solution into a vein of a limb which has been made ischæmic by a tourniquet. Most useful for operations on arms.

After suitable premedication preferably including a barbiturate to counteract any toxic effects of the local analgesia a needle with stylet in place is inserted into a suitable vein e.g. a vein in the cubital fossa. The limb is then elevated to empty it of blood and an Esmarch bandage is applied from the finger tips to a point at least 2 in. above the operation site. A second Esmarch bandage is now applied as a tourniquet proximal to the first one which is then unwound from above downwards. A third Esmarch bandage is applied as a venous tourniquet below the operation site; this is omitted by some workers.

The stylet is removed from the intravenous needle and 0.5 per cent solution of procaine is injected. Some force may be required for this. For an arm the dose is 30–60 c.c. for a leg 50–100 c.c.

If the proximal bandage causes discomfort an intradermal and subcutaneous band of analgesic solution injected above the bandage will remove the discomfort. Otherwise an Esmarch bandage is applied just distal to the painful

i.e. it is applied over an anæsthetized part of the limb the offending bandage can then be removed

Anæsthesia may not be complete for fifteen minutes but usually comes on more rapidly Muscular paralysis follows analgesia Analgesia lasts for two hours but disappears almost immediately after the proximal bandage is removed

The bandages must be put on carefully as analgesia will be imperfect if the limb is not absolutely ischæmic

### INTRA ARTERIAL LOCAL ANALGESIA

Introduced by Goyanes a Spaniard in 1912

Technique similar to intravenous local analgesia Seldom used

## CHAPTER XVIII

# THE USE OF MUSCLE RELAXANTS IN ANÆSTHESIA

## DEXTRO TUBOCURARINE CHLORIDE

It is usual to classify muscle relaxants used in anæsthesia as (1) Non-depolarizing (or anti-depolarizing) agents e.g. *d* tubocurarine gallamine triethiodide laudexium methylsulphate benzquinonium chloride and (2) Depolarizing agents e.g. decamethonium suxamethonium and suxethonium halides Under certain circumstances the depolarizing drugs can exert an anti-depolarizing effect the so-called mixed or biphasic block Other factors have to be considered such as the ionic cell environment the tissue blood flow and the interaction together of different drugs

*D* tubocurarine is generally supplied in 1.5 ml ampoules containing 15 mg and this is usually miscible with solutions of thiobarbiturates The alkaloid is a quaternary ammonium compound Tubadil is a suspension of *d* tubocurarine in oil 25 mg per ml and is said to have an effect lasting up to twenty four hours It is used to relieve pain due to muscular spasm e.g. after hæmorrhoidectomy

It is advised that a uniform strength of solution should be used i.e. 3 mg per ml It is simple to add 1.5 ml of *d* tubocurarine solution to 3.5 ml of saline in a 5 ml syringe which gives the recommended strength

**Source of Curare**—From the bark leaves and vines of the tropical plant *Chondrodendron tomentosum* growing near the upper reaches of the Amazon Has long been used by natives of S. America to poison the heads of their arrows They transport it in bamboo tubes hence the name tubocurarine

**History of Curare**—

1825 Introduced into Europe by Charles Waterton

1840 Claude Bernard proved that it acted by paralyzing the myoneural junction



**d Tubocurarine Chloride—History of Curare continued**

- 1802 Curare used by Chi holm in the American Civil War  
 1912 Used to produce relaxation of muscles by Laewen of Zwickau during surgery  
 1935 King isolated d tubocurarine chloride from the crude drug Ranyard West used it in tetanus  
 1938 Gill popularized its use in the U.S.A  
 1939 H Palmer used it for E.C.T. in England  
 1940 Bennett used it to soften the convulsions produced iatrogenically in psychiatry  
 1942 Griffith and Johnson used it to produce relaxation in anaesthesia at the suggestion of Wright on Jan 23  
 Gray and his colleagues (1946) have been pioneers of its use in Britain

**The Physiology of Muscular Contraction.**—It was shown by Sir Henry Dale years ago that a motor nerve liberates acetylcholine at the myoneural junction on the arrival of a nerve impulse. The motor end plate is specifically sensitive to acetylcholine and becomes depolarized (made electrically negative) by it (the motor end plate potential). This current of depolarization excites the adjacent part of the muscle fibre (muscle action potential) and passes along the membrane of the muscle fibre and is the final stimulus for causing the contraction of the contractile part of the muscle fibre. The released acetylcholine is meanwhile hydrolysed by the true acetylcholinesterase in the region of the motor end plate the two substances combining together so that when the excited muscle fibre has come out of its refractory state it will not become excited again by a depolarized end plate unless a new nerve impulse has arrived and released a new supply of acetylcholine.

**Neuromuscular Block.**—Drugs which cause neuromuscular block are described as —

- 1 Non depolarizers of the myoneural junction
- 2 Depolarizers of the myoneural junction
- 3 Agents which interfere with the formation of acetylcholine e.g. procaine excess of magnesium and phosphate ions deficiency of calcium ions the toxin of the *B. botulinus*. This third group of drugs is not of great importance to anaesthetists.

Neuromuscular blocking agents compete with acetylcholine for cholinergic receptors at the end plate and prevent the access of acetylcholine to these receptors. After adsorption to the receptor protein the depolarizing and non-depolarizing drugs behave differently.

- 1 The non-depolarizing agents (formerly called substitution blockers or competitive inhibitors of acetylcholine e.g. d tubocurarine gallamine laudexium) prevent access of acetylcholine to the receptor protein so that no depolarization no change in resting potential of the motor end plate and no muscular contraction—with consequent paralysis—take place. The paralysis is increased by additional non-depolarizing drugs by ether cyclopropane and procaine and is

- decreased by anticholinesterase drugs (e.g. neostigmine) depolarizing drugs potassium and calcium ions and adrenaline
- 2 The depolarizing drugs (e.g. decamethonium and suxamethonium) cause prolonged depolarization of the end plate like that caused by acetylcholine which in this case persists. The depolarization spreads to the muscle fibres making them electrically unresponsive to subsequent stimuli even after repolarization is complete. The repolarization phase of normal neuromuscular transmission is interfered with. Flaccid paralysis preceded by transient stimulation results in normal man (but not in myasthenic man and in some other species). The paralysis is increased by additional depolarizing drugs and by anticholinesterases. It is decreased by non-depolarizing drugs magnesium and by ether and cyclopropane.

**Dual, Biphasic or Mixed Block.**—While a single dose of a depolarizing drug causes pure depolarizing block (except in myasthenia when it causes a mixed block at the first injection) repeated doses can cause a change in response of the motor end plate so that non depolarizing block follows the original depolarizing block. If this dual block is present it is of course increased by non depolarizing drugs and decreased by anticholinesterases e.g. edrophonium. Further developments in this interesting branch of physiology are likely to take place and will eventually link up with the aetiology of myasthenia gravis.

**Pharmacology**—The drug has neither anæsthetic nor analgesic properties when given in clinical doses.

**EFFECT ON MOTOR END PLATE**—It is the classical non depolarizing myoneural blocking agent and acts by preventing the adsorption of acetylcholine to the cholinergic receptors and so prevents the changes in the end plate which cause muscular tone and contraction. Therapeutic doses produce the following effects in sequence: ptosis; imbalance of extra ocular muscles with diplopia (which may last several days); relaxation of muscles of the face, jaw, neck and limbs; relaxation of the anterior abdominal wall muscles; The intercostals and the diaphragm are the last muscles to be paralysed, their weakness giving rise to diminished respiratory movements and finally to apnoea. Muscular power returns in from fifteen to fifty minutes.

**CEREBRAL EFFECTS**—None in clinical doses. The electrical activity of the brain is first increased later decreased. Intracerebral injection into animals has caused convulsions. Large doses have been suspected of causing depression of the medullary centres and this may be one cause of prolonged apnoea following its use \* especially if complicated by depletion of intracellular potassium.

**EFFECTS ON AUTONOMIC NERVES**—These are due to substrate competition with acetylcholine for cholinergic receptors of autonomic ganglia and result in autonomic block without preliminary ganglionic stimulation. The block is not complete.

**d Tubocurarine Chloride—Pharmacology continued**

but diminishes vascular tone and may help to prevent shock. The alkaloid has a greater effect on sympathetic ganglia than the other relaxant, and a greater effect on pre than on post ganglionic neurones.

**EFFECTS ON RESPIRATION**—It paralyzes the muscles of respiration, the diaphragm being less sensitive than other muscles, is usually the last one to be paralysed. Bronchospasm, perhaps due to histamine release (aided sometimes by the barbiturates) is sometimes seen, especially in patients subject to asthma who are subjected to upper respiratory tract stimulation. Usually pharyngeal, laryngeal and bronchial reflexes are depressed so that endotracheal intubation and irritant anæsthetic vapours are well tolerated.

**EFFECTS ON CIRCULATION**—In ordinary doses there is no effect on the heart. There may be slight hypotension due to sympathetic ganglionic blockade together with histamine release. The clotting time is not altered. There is an antifibrillatory action on the ventricles and a decreased likelihood of arrhythmia. Hypertension (not due to hypercapnia) has also been reported.

**GASTRO-INTESTINAL SYSTEM**—The effect on the stomach and intestine is variable. The cardiac sphincter is probably not relaxed.

**ABSORPTION AND EXCRETION**—Absorbed when administered intravenously, intramuscularly, subcutaneously, intraperitoneally, sublingually and per rectum. In practice nearly always given intravenously, occasionally intramuscularly. It is excreted via the kidneys, some of it being first destroyed by the liver.

**OTHER ACTIONS**—It has been recommended for the control of certain reflexes which give rise to hypotension and bradycardia, e.g. carotid sinus, coeliac plexus, pelvic reflexes. It acts in these cases by depressing parasympathetic ganglia. It releases histamine from the tissues and heparin from the liver. It crosses the placental barrier but only in small amounts and does not affect the foetus clinically when the dosage is reasonable. It may increase the uterine tone. Patients with liver disease may require more *d* tubocurarine than usual to compete with the increased amount of acetylcholine resulting from the poor supply of pseudocholinesterase.

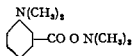
After intravenous injection effect commences within ten seconds and is at its maximum within four minutes. After intramuscular injection effect is seen after twenty minutes. After oral administration it is inactive unless taken in excessive dosage.

Even moderate dosage causes depressed breathing or apnoea and usually demands assisted or controlled respiration for its relief.

**Antagonists to Non depolarizing Relaxants—**

**NEOSTIGMINE**—The clinical antagonist is neostigmine methyl sulphate (prostigmine) which was synthesized by Aeschlimann

and Reinert in 1931 and which is twice as powerful as physostigmine the anticholinergic action of which was discovered by Pal in 1900. Neostigmine is an anticholinesterase. D-tubocurarine combines with the receptor protein without causing depolarization and thereby prevents acetylcholine from combining with the receptor and causing depolarization. If now the acetylcholine can be raised by increasing the amount of anticholinesterase (e.g. neostigmine) present to a level at which the excitation threshold of the end plate is reached then neuromuscular block will be overcome.



Neostigmine besides being an anticholinesterase is also a depolarizer and can cause on its own a depolarizing type of block but the amount needed to cause paralysis by persistent depolarization is much greater normally than that required to antagonize an effective dose of d-tubocurarine or gallamine. In addition it is a direct stimulant of cholinergic effector cells and has a direct stimulant effect on muscle. It has nicotinic effects i.e. in small doses it stimulates and in larger doses it depresses autonomic ganglia and has muscarinic effects which are blocked by atropine e.g. bradycardia, intestinal peristalsis and spasm, bronchial and salivary secretion and bronchospasm. It induces menstruation in certain cases of delay and may cause decreased coronary flow. It potentiates morphine analgesia. It is probably destroyed in the liver and excreted by the kidneys. Dose 0.5 mg carefully repeated up to a total of 5 mg preceded by atropine gr  $\frac{1}{16}$  (1.3 mg).

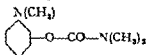
**EDROPHONIUM (tensilon)**—This is 3-hydroxy phenyl dimethyl ethyl ammonium chloride and is supplied in 1 per cent solution. It has like neostigmine anticholinesterase, depolarizing and direct stimulating effects on the motor end plate. Muscarinic effects are present but are not as severe as those of neostigmine; nevertheless atropine is required to combat them. Its actions come on quicker but do not last as long as those of neostigmine; consequently hypoventilation and recurarization may follow an initial stimulant effect on the respiration. Dose is 10 mg carefully repeated; this is equivalent in immediate effect to 1.25 mg of neostigmine.



**PYRIDOSTIGMINE (mestinon)** is the dimethyl carbamate ester of N-methyl pyridium bromide. It has half the potency of

## Antagonists to Non depolarizing Relaxants—Pyridostigmine continued

neostigmine but a longer duration of action. It is not as reliable as neostigmine as an antagonist to d-tubocurarine. Dose 5 mg



These three drugs are used also in the diagnosis and treatment of myasthenia gravis

### Clinical Uses —

- 2 They should not be given until some spontaneous respiration has returned

The muscarinic effects especially bradycardia must be prevented by atropine (gr  $\frac{1}{4}$  (1.3 mg) for each 2.5 mg) which can be given before with or after the neostigmine—good reasons are advanced for each of these times. The author at present prefers to cause mild tachycardia with atropine before injecting the neostigmine.

- 3 The maximal dose should probably seldom exceed 5 mg as neostigmine can itself cause myoneural block of a depolarizing type

- 4 As both gallamine and to a less extent dextro-tubocurarine block the vagal ganglia neostigmine is safer in curarized patients than in those with respiratory depression due to pethidine or thiopentone

- 5 Cyclopropane thiopentone jaundice and duodenal ulcer may all be associated with vagotonia and so are potentially dangerous if neostigmine is used

- 6 Neostigmine may cause bronchial constriction and secretion and so may predispose to atelectasis

- 7 Edrophonium is not a very satisfactory antidote to curarizing agents. It acts quickly but only for a short time so that recurarization has been seen. About 20 mg equals 2.5 mg of neostigmine.

Adrenaline, potassium calcium and guanidine are antagonistic to curare but are not used clinically

Signs of decararization after injection of neostigmine for inadequate respiration —

- 1 Return of normal tidal exchange measured either by an anemometer or by the flow meters of the anaesthetic machine using a Ryan or a Ruben valve
- 2 Effective coughing on endotracheal tube
- 3 Ability to open eyes and keep them open

### Signs of incomplete decyranization --

- 1 Persistence of chin or tracheal tug (sometimes) this may be associated with intercostal paralysis
- 2 Ineffective coughing on tube
- 3 Ptosis
- 4 General fidgeting jerky movements

Deaths have occurred following the use of neostigmine and atropine possible causes are (1) Cardiac inhibition (2) Adrenergic effects of neostigmine and atropine causing ventricular fibrillation (3) The atropine itself causing tachycardia especially in the presence of hypercapnia \* The author does not use neostigmine routinely but in many clinics it is employed in almost every patient who has received a non-depolarizing myoneural relaxant. It is however of fundamental importance that the patient should not be left until he is able to ventilate himself adequately.

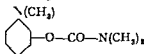
#### Clinical Uses of *d* Tubocurarine Chloride —

- 1 To make endotracheal intubation by the direct route relatively easy and atraumatic by shortening the duration of laryngeal spasm
- 2 To aid muscular relaxation during light anaesthesia
- 3 To lessen laryngeal spasm and quieten upper respiratory reflexes during anaesthesia
- 4 To facilitate control of respiration in thoracic and upper abdominal surgery
- 5 To facilitate bronchoscopy oesophagoscopy etc
- 6 To reduce the intensity of muscular contractions in electroplexy

**Technique** — Because of the occasional patient who is susceptible to the drug Gray advises that a trial dose should be given intravenously to the conscious patient and its effects watched. He gives 5 mg. and waits four minutes. The normal response of this dosage may be lassitude and difficulty in keeping the eyes open with some diplopia. The susceptible patient will show ptosis not under voluntary control dysphagia dysarthria and difficulty in breathing. Development of any of these signs calls for a reduction of subsequent dosage.

Following this trial injection in the ordinary abdominal operation a further 10–25 mg. of *d* tubocurarine chloride is injected and is followed immediately by the injection into the same needle of 4 to 10 ml. of 2½ per cent thiopentone solution. The face mask is now applied and a gas-oxygen mixture is gently forced into the lung (during inspiration if the patient is breathing). After a minute or two the larynx is exposed, sprayed with a topical analgesic solution and intubated under direct vision through a laryngoscope. Assisted or controlled breathing is maintained and additional doses of thiopentone perhaps pethidine and *d* tubocurarine are given as required. Gas-oxygen alone can be used. Gas-oxygen pethidine (in serial doses intravenously) gas-oxygen with minimal trilene is useful as an additional laryngeal sedative to prevent the patient coughing on the endotracheal tube. cyclopropane can be given or gas-oxygen-ether. If ether is used with *d* tubocurarine the dose of the latter must be considerably less than if thiopentone cyclopropane trilene etc. are used. This is because of the curarizing effect of ether.

**Antagonists to Non depolarizing Relaxants—Pyridostigmine** *continued*  
 neostigmine but a longer duration of action. It is not as reliable as neostigmine as an antagonist to d-tubocurarine. Dose 5 mg.



These three drugs are used also in the diagnosis and treatment of myasthenia gravis.

### Clinical Uses —

- 1 They should not be given until some spontaneous respiration has returned
- 2 The muscarinic effects especially bradycardia must be prevented by atropine (gr  $\frac{1}{2}$  (1.3 mg) for each 2.5 mg) which can be given before with or after the neostigmine—good reasons are advanced for each of these times. The author at present prefers to cause mild tachycardia with atropine before injecting the neostigmine
- 3 The maximal dose should probably seldom exceed 5 mg as neostigmine can itself cause myoneural block of a depolarizing type
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- 3 Ptosis
- 4 General fidgeting jerky movements

*d* tubocurarine and inflation with gas and oxygen intubation with a cuffed tube and controlled breathing. No more thiopentone is ordinarily given but additional doses of relaxant are freely used to provide proper operating conditions e.g. to abolish returning respiratory movements, returning muscle tone, reflex response, hiccups etc. Neo-stigmine and atropine at the end of the operation enable normal respiration to be resumed before the patient leaves the operating room.

The light levels of general anaesthesia used with the relaxants are not accompanied by vasomotor depression hence shock is lessened.

Since the use of the curare drugs became general the need for endotracheal intubation has decreased. The average lower abdominal operation of moderate duration should seldom call for intubation as reflex laryngeal spasm is rare, always provided that the stomach and oesophagus contain no vomitable material. Assisted or controlled respiration can be performed without an endotracheal tube and inflation of the stomach with anaesthetic gases can be prevented by maintaining a clear airway and avoiding excessive inflation pressure. Many anaesthetists of experience however intubate all their abdominal cases.

Cæsarean sections have been successfully performed under light general anaesthesia with *d* tubocurarine. Reports state that while the uterus retracts actively the baby is born crying vigorously. The relaxant does not readily pass the placental barrier.

Infants and children tolerate the drug well if the dosage is suitably adjusted to their general condition and body weight. An average dosage is 2 mg per stone. Not suitable for neonates.

The drug has been used without general anaesthesia in small dose for cataract extraction. It has an early and selective action on the extra-ocular muscles when given in small doses.

Dosage bears no relationship to age, sex or weight.

**Advantages and Disadvantages**—With this drug and its allies profound muscular relaxation can be produced quickly while the patient is under very light general anaesthesia. This combination of relaxant and light anaesthesia is relatively non-toxic while it does not add to operative shock. The lissive effects on the muscles of the larynx and bronchi remove the worrying symptoms of spasm of these tubes which have marred so many otherwise smooth anaesthetics.

Reduced tidal exchange or frank apnoea may readily be produced and unless remedied will seriously harm the patient. Emphasis must be placed on the danger of the patient returning to bed with a poor tidal exchange which will not only cause oxygen lack and carbon-dioxide excess but will also predispose to the formation of patches of collapse of the lungs. Inspiratory stridor due to atony of the vocal cords has been described following the use of relaxants.



*d* Tubocurarine Chloride—Technique continued

The inhalation agent or agents can be given in the closed circuit (if trilene is not used) or in the semi open semi closed circuit. If the latter is used a Ruben or a Salt expiratory valve makes assisted or controlled breathing easy and efficient. These aids to proper interchange of gases must always be used if the tidal exchange is reduced in volume a very frequent occurrence when proper abdominal relaxation is produced by *d* tubocurarine chloride.

During thiopentone gas-oxygen *d* tubocurarine anaesthesia the following signs often indicate the need for extra relaxant —

- 1 Rigidity of the abdominal wall
- 2 Increased amplitude of respiration
- 3 Bucking or coughing on the endotracheal tube
- 4 Increased resistance to inflation of the lung (in the absence of respiratory obstruction)

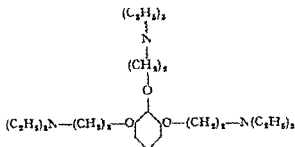
Extra analgesic is required if (1) The patient moves his skeletal muscles in response to the surgical stimulus e.g. face limb or neck muscles (2) The pulse rate rises

Whenever muscle relaxants are used it is of paramount importance to see that the patient is breathing reasonably deeply before he leaves the operating table. Nice judgement is often needed as to how much drug is required to make the peritoneum loose for the closure of the wound and when it should be injected without causing depressed breathing when the last skin suture is tied. Respiratory depression at the end of the operation should be treated either by inflation until the tidal volume becomes normal or by the injection of neostigmine with atropine. In thoracic cases it should be used routinely.

**OTHER METHODS** —There are many other techniques of using the muscle relaxants and the following may be mentioned —

- 1 Induction and maintenance with an intravenous barbiturate together with inhalation of gas-oxygen. Additions of relaxant just before skin incision and again if necessary before the abdomen is explored with further additions as required.
- 2 Maintenance with cyclopropane with relaxant given when required.
- 3 Maintenance with gas oxygen ether with relaxant given as required in smaller dosage than average.
- 4 Maintenance with a general anaesthetic with injection of a large dose (in several separate amounts) sufficient to produce apnoea. This must be followed by controlled breathing—a method useful in thoracic and upper abdominal operations.
- 5 Induction with thiopentone (up to 0.5 g in 2½ per cent solution) followed by gas and not less than 25 per cent oxygen with a muscle relaxant as required (flaxedil finds a use here) and repeated small doses of pethidine e.g. 25 mg intravenously.
- 6 Among others the Liverpool school of anaesthetists rely on an initial dose of thiopentone for induction followed by

and Boué in France and by Mushin and others (*Lancet* 1949 **1** 726)  
Chemically it is tri ( $\beta$  diethylamonoethoxy) benzene triethyl iodide



**Physical and Chemical Properties**—Before solution it is a white amorphous powder with a melting point of 145–150° C. Dissolves in water and alcohol to form stable solutions compatible with thiopentone. Supplied in 4 per cent solution in ampoules containing 2 ml (80 mg) and 3 ml (120 mg) (2 per cent solution is used in France). Estimates vary as to its relative strength compared with 15 mg of *d* tubocurarine chloride. 80 mg is perhaps a fair estimate.

**Pharmacology**—Duration of effect shorter than that of *d* tubocurarine (20–30 minutes against 30–45 minutes). Gallamine has a curari form action on the neuromuscular mechanism (i.e. it interferes with the action of acetylcholine) and previous doses of *d* tubocurarine chloride sensitize the patient to it. Its action is antagonized by the anticholinesterases (e.g. neostigmine) more efficiently than is *d* tubocurarine. It has less effect on the sympathetic ganglia than *d* tubocurarine chloride (and so can be used with fluothane) but shows an atropine-like vagal blocking effect which results in tachycardia even with small doses e.g. 20 mg. This outlasts the relaxant effect. In some patients receiving cyclopropane this tachycardia is absent because of the action of the gas on the sino auricular node and autonomic conductive tissues of the heart. It sometimes causes a rise in blood pressure. Neostigmine rapidly reverses the tachycardia. It is said to be less liable to release histamine than *d* tubocurarine. It depresses the respiratory function and will produce apnoea if dosage is pushed. Allergic reactions have been reported following its use. It passes the placental barrier and perhaps should not be used in obstetrics. Excretion is by the kidneys and it should be used carefully in cases of renal impairment in case prolonged curarization results.

**Clinical Uses**—Similar to those of *d* tubocurarine chloride. 70 mg being the equivalent of 3 mg of *d* tubocurarine. Given intravenously the first dose usually loses its effect in half an hour. To relax the peritoneum in an average patient a dose of from 60 mg. to 160 mg. is needed. Children tolerate larger relative

**D Tubocurarine Chloride—Advantages and Disadvantages continued**

Occasionally some residual paresis of the muscles of accommodation persists for twenty four hours after operation making reading difficult

The drug must be used with special care if it is given to the same patient on two occasions within twenty four hours as a cumulative effect may occur

It must not be used in patients suffering from myasthenia gravis except for purposes of diagnosis when minute doses are given

It potentiates the hypotension caused by halothane so the two drugs are better not used together

So potent is *d* tubocurarine chloride and so likely is it to cause respiratory depression that its use should be restricted to experienced anaesthetists who are fully competent to institute and maintain efficient artificial respiration in all types of patient

In cases of liver damage Dundee and Gray\* have experienced abnormal resistances to *d* tubocurarine This may be associated with a lowered serum cholinesterase and increased sensitivity to acetylcholine at the end plate

**Other Uses of *d* Tubocurarine**—It has been used in the treatment of tetanus spasticity and rigidity associated with neurological disease orthopaedic conditions myositis arthritis and strychnine poisoning Unfortunately voluntary power is abolished as well as spasticity Tremor is not improved

As a diagnostic test for myasthenia gravis 0.1 to 0.2 mg per stone is slowly injected intravenously If myasthenia is present a marked exaggeration of its symptoms is produced in a minute or two These can be dissipated by the immediate intravenous injection of neostigmine and atropine (1.5 mg and  $\frac{1}{16}$  gr)

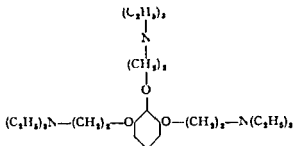
*Electric convulsion therapy* was formerly modified by the following technique Thiopentone *d* tubocurarine and atropine are all mixed in the same syringe and injected slowly dose of *d* tubocurarine 2.5 mg per stone in females 3 mg per stone in males dose of thiopentone 0.15 g in females 0.25 g in males dose of atropine  $\frac{1}{16}$  gr in both males and females Dosage is altered in subsequent treatments if necessary

After injection an airway is inserted and oxygen insufflated from a reservoir bag or resuscitation apparatus The convulsion is given two or three minutes after the injection and there after oxygen insufflation followed by inhalation is kept up as long as necessary Apnoea lasts from two to forty five minutes average being ten minutes The shorter acting relaxants are now commonly used to soften the convulsions of E.C.T

**GALLAMINE TRIETHIODIDE (Flaxedil)**

A synthetic curarizing agent producing non-depolarizing neuromuscular block first prepared by the Frenchmen Bovet and Halpern in 1947 First clinical report on its use in anaesthesia by Huguenard

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its action extends to the muscle-fibres themselves on each side of the end plate and makes them unexcitable. It acts like acetylcholine which cannot be hydrolysed. It prevents the access of acetylcholine to the cholinergic receptors of the end plate. Muscular twitching may be seen soon after its injection, an effect made worse by a high blood adrenaline level. It differs from *d* tubocurarine in liberating less histamine, in being more evanescent in action and in causing less paralysis of autonomic ganglia. It is not antidoted by the anticholinesterases such as neostigmine (which by causing acetylcholine to persist may increase the paralyzing effect of decamethonium) but by the compound pentamethonium iodide when given in 10-100 times the dose of decamethonium iodide. This effect is too dangerous to make clinical use of because of the resulting hypotension. Repeated use may lead to the depolarizing effect at the end plate giving place to a non-depolarizing effect, the so-called dual block and this if present can be reversed by edrophonium or neostigmine. This explains the tachyphylaxis seen on repeated dosage—one is really giving a depolarizing drug to a patient whose myoneural junctions are depressed by non-depolarization and this creates an antagonism. Patients with myasthenia gravis are said to be less affected by the drug than are normal people and a single injection results in dual block which is antagonized by edrophonium and neostigmine. Its relaxant effect lasts rather less time than that of gallamine (15-20 minutes). Either anaesthesia together with the presence of adrenaline in the circulation tends to lessen its action—the opposite effect to that seen when curariform drugs are used. Its action on muscles varies with the amount of blood circulating in them. It may dilate the pupils. Excreted largely in urine and not hydrolysed—hence its longer duration of effect than is seen with suxamethonium.

**Clinical Uses \*—**The drug causes a depolarizing block which is active but does not last long. It gives place to a non-depolarizing block which although incomplete is longer lasting. With increasing doses the non-depolarizing block becomes more complete. This as in the case of *d* tubocurarine reduces the depolarizing action of subsequent doses and may be the explanation of the tachyphylaxis seen after repeated injections. The average dose to relax the abdomen is 3 mg. which can be taken as the equivalent of 15 mg. of *d* tubocurarine chloride. A dose of 4-5 mg. will produce apnoea for 10 or 20 minutes. For abdominal surgery repeat doses may be required in 10 to 40 minutes after the initial dose. If sufficient is given to enable an easy closure of the peritoneum there is a good chance that the patient will be moving a reasonable tidal exchange at the end of the operation. It can be used to facilitate intubation when combined with thiopentone but is not a good inhibitor of the cough reflex making the drug unreliable in thoracic surgery. The patient may pass from apnoea to breathing disturbed by cough very rapidly—a highly inconvenient occurrence. In other cases prolonged apnoea has been

**Gallamine Triethiodide—Clinical Uses continued**

doses e.g. 1 mg per lb. Very useful for direct vision intubation (40–80 mg). An excellent drug which the author uses almost to the exclusion of *d* tubocurarine. More accurate doses can be given if it is diluted with saline to 2 per cent so that each ml contains 20 mg. Can be used intramuscularly with hyaluronidase (the ampoule of powder dissolved in the gallamine solution) in doses about 50 per cent greater than would be required by the intravenous route.

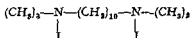
It is possibly better avoided (1) When there is pre-existing tachycardia (2) When there is renal impairment (3) In obstetrics

**DIMETHYL *d* TUBOCURARINE IODIDE  
(METUBINE, DME)\* BROMIDE (DIAMETHINE)  
AND CHLORIDE (MECOSTRIN)**

Prepared by King and Dutcher (1935). Pharmacological properties described by Collier in 1948. This non-depolarizing relaxant is 2–2½ times as potent as *d* tubocurarine; its action does not last as long and there is less histamine release and autonomic blocking action than is seen after *d* tubocurarine. Its onset is more rapid than and its duration half that of *d* tubocurarine and it is more sparing of the diaphragm. The average paralysing dose is 1 mg per stone and it is said to be often possible to maintain adequate spontaneous respiration together with good relaxation at least in lower abdominal operations. Prepared in 3 ml ampoules 2 mg per ml. It is said to enable inflation of the lung to be performed more easily than when other relaxants are used.

**DECAMETHONIUM†**

This is bistrimethylammonium decane dihalide and was originally known as C 10. It was introduced by Barlow and Ing and by Paton and Zaunis in 1948 and used in anaesthesia by Organe in the following year. Prepared commercially as Syncurine decamethonium dibromide and Eulissin decamethonium diiodide each ml of solution containing 2 mg.



**Chemistry**—In its pure form is a white crystalline salt soluble in water, neutral in solution, stable and resistant to heat. The solution mixes well with thiopentone and alkaloids and is non-irritant to tissues.

**Pharmacology**—It causes neuromuscular block by prolonged depolarization of the motor end plate in skeletal muscles, the intercostals and diaphragm being affected less intensely. In addition

See articles by H. B. Wilson, H. E. Gordon and A. W. Raffan *Lancet* 1950 **1** 1296 and Bullough, J. *Proc. World Congress Anaesthesiologists* 1956 251. Minneapolis: Burgess Publ. Co.

† See articles by G. Organe *Canad. Anaest. Soc. J.* 1956 **3** 5 and by A. R. Hunter *Brit. J. Anaesth.* 1950 **22**, 218.

its action extends to the muscle-fibres themselves on each side of the end plate and makes them unexcitable. It acts like acetylcholine which cannot be hydrolysed. It prevents the access of acetylcholine to the cholinergic receptors of the end plate. Muscular twitching may be seen soon after its injection, an effect made worse by a high blood adrenaline level. It differs from *d* tubocurarine in liberating less histamine, in being more evanescent in action, and in causing less paralysis of autonomic ganglia. It is not antidoted by the anticholinesterases such as neostigmine (which by causing acetylcholine to persist may increase the paralyzing effect of decamethonium) but by the compound pentamethonium iodide when given in 10-100 times the dose of decamethonium iodide. This effect is too dangerous to make clinical use of because of the resulting hypotension. Repeated use may lead to the depolarizing effect at the end plate giving place to a non-depolarizing effect, the so-called dual block, and this if present can be reversed by edrophonium or neostigmine. This explains the tachyphylaxis seen on repeated dosage—one is really giving a depolarizing drug to a patient whose myoneural junctions are depressed by non-depolarization and this creates an antagonism. Patients with myasthenia gravis are said to be less affected by the drug than are normal people and a single injection results in dual block which is antagonized by edrophonium and neostigmine. Its relaxant effect lasts rather less time than that of gallamine (15-20 minutes). Ether anaesthesia together with the presence of adrenaline in the circulation tends to lessen its action—the opposite effect to that seen when curariform drugs are used. Its action on muscles varies with the amount of blood circulating in them. It may dilate the pupils. Excreted largely in urine and not hydrolysed—hence its longer duration of effect than is seen with suxamethonium.

**Clinical Uses** \*—The drug causes a depolarizing block which is active but does not last long. It gives place to a non-depolarizing block which although incomplete is longer lasting. With increasing doses the non-depolarizing block becomes more complete. This as in the case of *d* tubocurarine reduces the depolarizing action of subsequent doses and may be the explanation of the tachyphylaxis seen after repeated injections. The average dose to relax the abdomen is 3 mg. which can be taken as the equivalent of 15 mg. of *d* tubocurarine chloride. A dose of 4-5 mg. will produce apnoea for 10 or 20 minutes. For abdominal surgery repeat doses may be required in 10 to 40 minutes after the initial dose. If sufficient is given to enable an easy closure of the peritoneum there is a good chance that the patient will be moving, a reasonable tidal exchange at the end of the operation. It can be used to facilitate intubation when combined with thiopentone but is not a good inhibitor of the cough reflex making the drug unreliable in thoracic surgery. The patient may pass from apnoea to breathing disturbed by cough very rapidly—a highly inconvenient occurrence. In other cases prolonged apnoea has been

\* Organe G. *Canad. Anaest. Soc. J.* 1956 3 5



**Decamethonium—Clinical Uses continued**

reported. On the cardiovascular system there is no apparent effect. No tendency to spasm of the bronchi has been reported. It is stated to have possibly some vagal inhibitory effect. It has been successfully used for Caesarean section and also to obtain relaxation in external version. It does not readily pass the placental barrier in spite of the small size of its molecule.

The drug should be confined to those operations expected to last less than ninety minutes and 10 mg should not be exceeded. If the operation is nearing its end *suxamethonium* 10–20 mg may be given and repeated. Otherwise it is better to finish the operation with gallamine 10 mg.

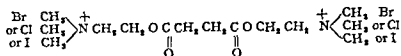
**LAUDERIUM METHYL SULPHATE (LAUDOLISSIN)**

This is a heterocyclic decamethylene bi quaternary ammonium compound synthesized by Taylor and Collier in 1951. It is a true non depolarizing myoneural blocking agent one half as potent as *d* tubocurarine, not always completely antagonized by neostigmine acting longer than *d* tubocurarine (45–50 minutes) and having no undesirable side effects. Its effects are potentiated by ether and antagonized by small doses of *suxamethonium*. It does not release histamine. Maximal effect comes on five minutes after injection. It is not miscible with thiopentone or pethidine, gallamine, atropine or *suxamethonium* chloride. A test dose of 10 mg should be given. (See articles by Bodman R I, Morton H J V and Wylie W D *Lancet* 1952 2: 517; Dundee J W, Gray T C and Riding J F *Brit J Anaesth* 1954 26: 13.)

**THE SHORT-ACTING MUSCLE RELAXANTS**

**History**—Prepared in 1906 by Hunt and Taveau. In 1949 Bovet and his colleagues described the paralysing action of the bis choline esters of succinic acid showing that they produced muscular paralysis of short duration and rapid onset. The first clinical use was reported from Sweden (Thesleff and von Dardel), from Austria (Brucke and Mayerhofer) and Italy in 1951 and in the same year Scurr published his experiences with a small sample of *suxethonium* iodide.

**Chemistry**—The active part of the molecule is the cation formed by the succinic radicle with a quaternary ammonium group at each end of the molecular chain. If these end groups contain three methyl groups ( $\text{CH}_3$ ) the substance is a *suxamethonium* compound; if two methyl and one ethyl ( $\text{C}_2\text{H}_5$ ) then it is an *ethonium* compound—hence *suxamethonium* and *suxethonium*. The cation is combined with one of the halides chlorine, bromine or iodine which forms the anion and this fact leads to the difference in the molecular weights in the different compounds. As the halide ion is inactive the cation identical in each case is the active moiety and its weight should be regarded as the weight of active drug used. Thus 100 mg of *suxamethonium* chloride contains 80 mg of active cation while 60 mg of *suxamethonium* bromide contains 40 mg of active cation. Deteriorates in hot climates.



### SUXAMETHONIUM HALIDES

The following compounds have been prepared

**SUXAMETHONIUM CHLORIDE** (succinylcholine chloride diacetylcholine succinyl dicholine chloride Anectine Lysthenon Paranoval Brevidil M (in solution) Quelcin Tachycuraryl Scoline) —The last is put up in 2 ml ampoules each containing 100 mg of the drug in 5 per cent solution equal to 40 mg of active cation per ml and in 10 ml bottles containing 500 mg of the drug or 400 mg of active cation

**SUXAMETHONIUM BROMIDE** (Brevidil M in powder) —Each ampoule contains 60 mg of dry powder equivalent to 40 mg of active cation

**SUXAMETHONIUM IODIDE** (Celocurine Succinylcholine iodide)

**SUXETHONIUM BROMIDE** (Brevidil E) —Each ampoule contains 150 mg of dry powder

**SUXETHONIUM IODIDE** (I S 362) —First prepared in Rome by Bovet in 1949

Suxamethonium solution is unstable when mixed with alkalis and loses activity when mixed with thiopentone

**Pharmacology** —These compounds like acetylcholine act by depolarizing the motor end plate. They prevent the access of acetylcholine to the cholinergic receptors of the end plate. They also influence the muscle fibres adjacent to the end plate making them unexcitable. This depolarization causes the fasciculation of muscle bundles especially in the neck and limbs seen after rapid injection. It causes post operative aching especially in ambulant patients and can be lessened if 10–20 mg of gallamine is injected before the suxamethonium (but there are pharmacological objections to this sequence)\*. Unlike acetylcholine however the drugs are not almost instantaneously hydrolysed so that depolarization remains and the muscle fibres become no longer electrically excitable and are in fact paralysed. The effects of suxamethonium are prolonged by hypercapnia and also by hypothermia.

Inactivation is by enzymatic hydrolysis caused by the pseudo cholinesterase of the plasma which converts the drug to succinic acid and choline both normal metabolites. This normally occurs in three to five minutes but very occasionally a prolonged effect with apnoea is seen and may be due to an abnormally low plasma pseudocholinesterase level†. Such a low level may be seen in states of malnutrition severe anaemia certain liver diseases and after contamination with agricultural poisons containing cholinesterase inhibitors. It must be realized that all anticholinesterase drugs e.g. neostigmine may cause a prolonged action and are absolutely contra indicated after the use of these depolarizing agents (except in the case of suspected

Churchill Davidson H C, *Br J Med Bull* 1958 14 1 31

† Fyfe I T Gray P W S, Lehm n H., and Silk E. *Lancet* 1952 1 1229

**Suxamethonium Halides—Pharmacology continued**

dual block when edrophonium 10 mg can be cautiously used) Neostigmine cannot be used to potentiate suxamethonium as the dose required would be excessive Should prolonged apnoea occur the plasma pseudocholinesterase level can be raised by the intravenous transfusion of whole blood preferably fresh but otherwise stored stored blood contains 30–50 units of pseudocholinesterase per ml A low pseudocholinesterase level is however only one cause of prolonged apnoea

Other causes of prolonged apnoea may be (1) The associated depressant drugs used (2) Acapnia from hyperventilation and hypercapnia from underventilation (3) Interference with the Hering Breuer reflex from distension of the lungs (4) Hypocalcaemia associated with muscular spasms (5) Incomplete hydrolysis the intermediate breakdown product succinyl monocholine has a more prolonged effect than its parent drug which is succinyl dicholine (6) It has recently been suggested that the normal breakdown product choline may itself perpetuate the block following the injection of large amounts of suxamethonium \* Heroic doses of nikethamide up to 20 ml intravenously have been suggested for its relief by Barron

There is antagonism between these compounds and the non depolarizing relaxants The drugs are not ganglionic blockers do not release histamine in any large amount clinically although wheal formation and bronchospasm have been described † and do not cross the placental barrier and are non irritating in solution They cause a slight rise in blood pressure Useful agents in kidney disease as they are hydrolysed in body and not excreted

On rapid intravenous injection there is muscular twitching especially noticed in the head and neck This very soon passes off and gives place to muscular relaxation which persists for three to five minutes and then in almost all cases rapidly passes off The suxethonium compounds have an even more evanescent action

Foldes and his colleagues (*Science* 1953 117 383) have demonstrated substrate competition for pseudocholinesterase between procaine (and xylocaine) and suxamethonium The use of procaine in conjunction with suxamethonium may cause delayed hydrolysis of the latter as both drugs appear to be hydrolysed by pseudocholinesterase Some of the drug may be excreted by the kidneys

**Cholinesterase**—Acetylcholine is an ester which is hydrolysed into acetic acid and choline by

- 1 True cholinesterase i.e. specific cholinesterase acetylcholinesterase red cell cholinesterase e type (erythrocyte) cholinesterase

**Pseudocholinesterase** i.e. non specific cholinesterase plasma or serum S type cholinesterase Free acetylcholinesterase is found in red cells and at cholinergic synapses and myoneural junctions—where it hydrolyses acetylcholine Pseudocholinesterase has little effect on acetylcholine at physiological concentrations but will hydrolyse other compounds Both enzymes are inhibited by neostigmine the anticholinesterase Pseudo cholinesterase is a mucoprotein formed in the liver and normally the serum contains 65–110 units A level less than 55 is low There is a high value in early childhood and as age advances it gets less Its amount is one measure of hepatic function

**Low pseudocholinesterase levels occur** (1) After therapeutic radiation (2) After contamination with organic phosphorus insecticides (3) In hyperpyrexia (4) In cardiac failure (5) In uraemia (6) In liver disease (7) In malnutrition (8) In severe anaemia (9) In hyperthyroidism (10) In very ill and handicapped patients

**Clinical Uses**—While suxamethonium is usually given by intravenous injection preferably in weak solution—1 per cent or less to minimize muscular fasciculations it can be injected intramuscularly in doses of 2 mg/kg or with the addition of hyalase in a dose of 1 mg/kg This may be useful in children before intubation

**FOR INTUBATION**—As the muscular twitches are quickly produced and painful the patient should first be put to sleep and then given the relaxant Suitable dose is 24–40 mg of active cation e.g. 40–50 mg of suxamethonium chloride To overcome acute laryngeal spasm 50 mg of suxamethonium should be injected followed by rapid intubation

**FOR ENDOSCOPY**—Serial doses are given as required under general anaesthesia e.g. by thiopentone the first before the introduction of the endoscope subsequent ones as required Oxygen should be given concurrently and respiration should not if possible be completely abolished

**FOR ORTHOPÆDIC MANIPULATIONS**—A short period of profound relaxation is produced together with a moderate dose of thiopentone

**FOR ABDOMINAL CLOSURE**—Doses can be given serially during the suture of the peritoneum with the full expectation that normal breathing will have returned as the skin stitches are completed Should the patient still be partly curarized from gallamine or *d* tubocurarine it is dangerous to give neostigmine at the end of the operation to completely overcome the effects of the former drugs neostigmine being a cholinesterase inhibitor and likely to delay hydrolysis of suxamethonium

**FOR ELECTROCONVULSIVE THERAPY** (*see later*)

Suxamethonium raises the intra-ocular pressure and should not be used during intra-ocular surgery Enophthalmos has been observed after the use of suxamethonium \*

Suxamethonium Halides—Clinical Uses *continued*

The *continuous suxamethonium drip* is used as a controllable and rapid method of maintaining relaxation of different degrees during abdominal and other surgical procedures. Various strengths have been described, a popular one containing 10 ml of suxamethonium chloride in 500 ml of saline giving a dilution of 0.1 per cent. If this amount of drug is added to 400 ml of saline then the strength is 0.1 per cent of active cation.

The solution is run in from a standard drip set at a rate of 30 drops per minute until muscle weakness develops. No twitching is seen at this slow rate and undue sensitivity to the drug is quickly noticed. Thiopentone is then injected (0.25–0.5 g) and the drip speeded up to 60 drops a minute. When the jaw is fully relaxed oxygen can be given and then the laryngoscope is used in the ordinary way. During maintenance of anaesthesia it is desirable in the eyes of some workers\* to keep respiratory movements from complete abolition; otherwise it is difficult to ascertain if the patient is receiving too much drug. Many other workers abolish respiration and allow it to return occasionally during the operation. Assisted or controlled breathing must of course be carried out. The amount used varies between 4 and 10 mg per minute of the cation. When the peritoneum is sutured the drip may be stopped. Average rate of drip 30 to 60 drops a minute or 150 to 400 ml each hour. If after a prolonged suxamethonium drip the patient is requiring increasing amounts of drug (i.e. is probably developing a mixed block) it may be better to give a small dose of a non-depolarizing relaxant to maintain block to avoid accumulation of succinyl monocholine. Such small doses last a long time and can be reversed by neostigmine.

- A disadvantage of the suxamethonium drip is that although respiration may return easily enough at the end of the operation it may be somewhat depressed in volume. Some workers prefer serial injections of 50 mg given at carefully judged intervals to the continuous drip.

**Prolonged Apnoea following Anaesthesia.**—This has occurred and has proved fatal following the use of non-depolarizing relaxants† depolarizing relaxants and the combination of the two. It has also been seen after intra and extra-dural blocks which have involved the phrenic roots presumably and in medical patients with chronic hypercapnia who have been given a high oxygen atmosphere to breathe. Streptomycin and neomycin potentiate myoneural block.

1. **PROLONGED APNŒA AFTER THE USE OF NON-DEPOLARIZING RELAXANTS†**—Evidence is accumulating that the non-depolarizing relaxants may have abnormal effects if the potassium metabolism is disordered as in dehydration and electrolyte depletion. One of the abnormal effects may be a

Foldes, F. F., *Muscle Relaxants in Anaesthesiology* 1957, 19. Springfield, C. C. Thomas.  
 † Hunter, A. R. *Brit med J* 1956 2, 970.  
 Foster, P. A. *Brit J Anaesth* 1956 28, 488.

central depressant action on the medullary centres leading to respiratory and circulatory failure. It may be associated with an initial resistance to the relaxant—depolarizing or non depolarizing. Non-depolarizing relaxants should be used most carefully in ill and handicapped patients and in those with electrolyte imbalance and intracellular potassium depletion e.g. in dehydration starvation vomiting diarrhoea metabolic disturbances acidosis diabetic coma and after the use of cortisone. The rapid intravenous injection of 0.3 per cent potassium chloride 100–200 ml (controlled by the E.C.G.) may be helpful. A normal blood potassium level does not exclude a reduced intracellular potassium content especially when dehydration is present. If reasonable doses of neostigmine fail to restore normal breathing 30–60 mg of ephedrine should be injected intramuscularly. This diminishes the rate of adrenaline destruction thereby potentiating neostigmine. Adrenaline has of course an anticholinergic action and also helps to maintain the high potassium level of the muscle fibre. In addition ephedrine helps to maintain the blood pressure.\*

## 2. PROLONGED APNŒA AFTER DEPOLARIZING RELAXANTS—This may be due —

- a To over-dosage with the relaxant drug. It is interesting to remember in this connexion that in order to prevent over dosage of suxamethonium Foldes† advises that with a continuous drip apnœa should never be allowed to occur. Assisted rather than controlled respiration is advised.
- b To a low pseudocholinesterase level in the blood. This seldom causes prolonged apnœa if 50 mg is not exceeded a dose adequate for most patients requiring a single injection e.g. for intubation or E.C.T. It is unlikely to be the cause of apnœa prolonged beyond 20–30 minutes. A low pseudocholinesterase level may occur in liver disease severe anæmia malnutrition starvation electrolyte imbalance thyrotoxicosis and contamination with those organic phosphorus insecticides which are pseudocholinesterase inhibitors and include hexaethyl tetraphosphate (HETP) tetraethyl pyrophosphate (TEPP) parathion pestox 3 isopestox.
- c To excessive formation of succinyl monocholine. In 1952 Whittaker and Wijesundera pointed out that the hydrolysis of succinylcholine takes place in two stages succinyl monocholine being the intermediate product. This has between  $\frac{1}{2}$  and  $\frac{1}{3}$  the relaxing effect of the parent compound but as it is hydrolysed rather slowly by both true and pseudo cholinesterase it may accumulate in the blood stream but only if relatively large amounts of suxamethonium (1.5–2 g) have been used e.g. as a drip infusion.
- d To dual or mixed block a phenomenon described by Zaimis‡ in 1953. According to this theory the end plate can under

Burn J. H. *Brit J Anaesth* 1957 29 243  
 † Foldes F. F. *Muscle Relaxants and Anaesthesia* 1957 Springfield C. C. Thomas  
 ‡ Zaimis F. *J Physiol* 1953 122 238

**Suxamethonium Halides**—Prolonged Apnoea following Anæsthesia *contd*  
 certain circumstances react in such a way that the initial depolarizing block gives way to a non-depolarizing block which is antagonized by such anticholinesterase agents as edrophonium and neostigmine. Dual block is very rarely seen after single doses of either decamethonium or suxamethonium but may be the cause of confusion after repeated doses e.g. the suxamethonium drip or multiple doses of decamethonium. Should dual block be suspected as the cause of prolonged apnoea 10 mg of edrophonium should be injected. If the suspicion is correct respiration will re-start and can then be helped by additional edrophonium or neostigmine together of course by artificial respiration. If the cause is true prolonged depolarization then it will be potentiated but for only a short while by the evanescent edrophonium.  
 c To the myoneural blocking effect of the normal product of suxamethonium breakdown choline if large amounts of suxamethonium e.g. 400–500 mg have been injected \*.

#### **Other Causes of Prolonged Apnoea —**

- 1 Central depression of the respiratory centre by morphine or its congeners thiopentone cyclopropane
- 2 Hypocapnia in this case respiration will recommence if the blood carbon dioxide level is allowed to rise
- 3 Hypercapnia prolonged hypoventilation or deficient carbon dioxide elimination from the anæsthetic circuit can poison the respiratory centre and cause apnoea. It can sometimes be overcome by vigorous hyperventilation using either fresh soda lime or a non-rebreathing circuit
- 4 Depression of the Hering Breuer mechanism during controlled respiration. This will usually yield to more gentle inflation together with the addition of a little carbon dioxide for short periods
- 5 Reflex laryngeal apnoea. Due to the presence of an endotracheal tube. Removal of the tube leads to restoration of spontaneous respiration

#### **Diagnosis of the Cause of Prolonged Apnoea —**

- 1 Electrical stimulation of a peripheral nerve to exclude neuromuscular block (for apparatus see Christie T H and Churchill Davidson H C *Lancet* 1958 **1** 776)
- 2 Examination of a blood specimen—taken without air contact—to assess blood CO<sub>2</sub> level and blood pseudocholinesterase level
- 3 Edrophonium to exclude dual block. See also Paton W D *Anæsthesia* 1958 **13** 253

#### **Differential Diagnosis between Apnoea due to Central Depression and that due to Peripheral Paralysis †—**

- a CENTRAL DEPRESSION—Breathing slow reasonably deep. No tracheal tug. no pause at end of inspiration
- b PERIPHERAL PARALYSIS (Myoneural Block)—Breathing jerky shallow and of normal rate. Pause after inspiration and again after expiration (Morton's rectangular breathing). Tracheal tug

**Use of Relaxants in Electroplexy**—Convulsions were first used in psychiatry in 1934 by Meduna. Cerletti and Bini inducing them electrically in 1938.

The stomach and bladder must be empty and the patient reassured. Thiopentone is given intravenously the dose varying from 0.2 to 0.4 g. Some workers give atropine gr  $\frac{1}{4}$  routinely others avoid it. The author prefers to give it combined with the thiopentone. Suxamethonium chloride 40–50 mg in 1 per cent solution (to reduce the incidence of post operative muscular pain) is now injected. When the twitching ceases the patient is ready for the attention of the psychiatrist. After the convulsion it may or may not be necessary to inflate the lungs with oxygen. In poor risk cases this can advantageously be done after the injection of the thiopentone. Damage to the teeth and lips is a real danger and is likely to occur during the passage of the current rather than during the modified convulsion. Inflation of the chest with oxygen during the convulsion prolongs it. After the current is passed a modified convulsion is likely if (1) There is a pilomotor reaction (2) The pupil fails to contract when inspected. When teeth are present they should be separated by firm rubber tubing or other suitable bite block.

Modifications of this technique include giving 50 mg of chlorpromazine by mouth two hours before the procedure as a tranquillizer (and a venous dilator!) substituting gallamine 30–60 mg for suxamethonium with or without the addition of neostigmine after the convulsion. Psychiatrists vary in the amount of paralysis they require and are like surgeons in this respect! Some believe that a certain amount of healthy jerking is beneficial to their patients and so smaller doses of relaxants (and greater risk of fractures) will be the order of the day.

As both suxamethonium and E.C.T. raise the blood pressure patients with hypertension or cardiac hypertrophy will need careful assessment before being subjected to this heroic but very effective form of treatment.

See articles by Edridge A. and Ferguson A. L. *Proc R Soc Med* 1952 45 12 and Adderley D. J. and Hamilton M. *Brit med J* 1953 1 195.

**Use of Relaxants in the Treatment of Tetanus**—All of the commonly used relaxants have been used to control the spasms of tetanus at one time or another and so has chlorpromazine.\*

Mephanesin given into the stomach by Ryle's tube because it is a local analgesic of the pharynx has been successfully used in mild cases. The initial dose should be 1 g given intravenously well diluted followed by one or two ounces of the elixir into the stomach and repeated as may be necessary. The drug can be given also intramuscularly.

The use of a continuous drip of suxamethonium shows great promise. Constant care is necessary while the rate should be increased.

Bodinan R. I., Morton H. J., and Thomas E. T. *Lancet* 1955 2 230 and Adrian J., and Kerr M. *Sib med J (Bham Ala)* 1955 48 858.



**Suxamethonium Halides—Relaxants in the Treatment of Tetanus** *continued*

before the patient receives such stimuli as intravenous injections suction etc (See Woolmer R and Cates J E *Lancet* 1952 2 808)

In severe convulsive tetanus the plan should be to induce flaccid paralysis and maintain artificial respiration. Tracheostomy should be considered thiopentone and gallamine being a good anæsthetic for this operation. The patient may well be connected to an artificial respirator and given air and oxygen mixture by intermittent positive pressure following the method used by Ibsen and Lassen during the epidemic of poliomyelitis in Denmark. Sedation can be achieved with gas-oxygen mixtures or with sodium amylobarbitone 200 mg qh. A cuffed tracheostomy tube will prevent soiling of the lower air passages. Suction may be necessary and the head-down tilt will help drainage from the chest. The patient will require frequent change of position antibiotics and daily chest radiographs. A suitable relaxant should be given as required. Central depressants are bad. chloral paraldehyde barbiturates and bromethol are all respiratory sedatives and should be given only to bring sleep (See articles by Shackleton P *Lancet* 1954 2 155 Honey G E Dwyer B E Smith A C and Spalding J M K *Brit med J* 1954 2 442 Galloway W H and Wilson H Bruce *Anæsthesia* 1955 10 303 Woolmer R *Brit med Bull* 1958 14 1 54)

**Use of Different Relaxants in the Same Patient**—The effects of *d* tubocurarine gallamine and laudexium are additive and so are those of decamethonium and suxamethonium. In general depolarizers should not be used after non-depolarizers. Only if the effects of the first drug have worn off should one of the other group be used. Before a depolarizing drug is able to relax muscles after a non-depolarizing drug has been given it first has to overcome the inhibition of the physiological depolarization caused by the non-depolarizing drug. Thus larger doses of a depolarizer are required to produce relaxation in a patient recovering from a non-depolarizing drug e.g. large doses of suxamethonium (i.e. at least 50 mg to 100 mg) must be given at the end of an operation for peritoneal closure in a patient recovering from *d* tubocurarine or gallamine and apnoea once achieved may be prolonged and difficult to reverse. It is a reasonable practice to use a single dose of suxamethonium for intubation followed by a non-depolarizing relaxant during the operation provided the effects of the suxamethonium are allowed to wear off before the non-depolarizing drug is given. Small doses of non-depolarizing relaxant give protection against subsequent doses of depolarizers. Prolonged administration of a depolarizing relaxant sensitizes the end plate to the effects of the non-depolarizers and decreases end plate sensitivity to additional depolarizer. A block caused by a small dose of a non-depolarizer given after prolonged administration of a depolarizer can be readily antagonized by a small dose of neostigmine.

**Use of Relaxants in Myasthenia Gravis —**

- 1 Non-depolarizing relaxants cause hypersensitivity of affected muscles only. Other muscles behave normally.
- 2 Decamethonium is abnormally well tolerated in mild cases but causes hypersensitivity in severe cases but the block is a non-depolarizing one preceded by a brief depolarizing block i.e. it is a dual or mixed block. Suxamethonium should be given very carefully.

**MEPHENESIN (MYANESIN)**

First described by Berger and Bradley in 1946 marketed by British Drug Houses. Chemically it is  $\alpha$ - $\beta$ -dihydroxy  $\gamma$  (2 methyl phenoxy) propane. Barnett Mallinson wrote the first clinical account of the drug in 1947. Known in the U.S.A. as Tolserol and Lissaphen and Atensin.

**Physical and Chemical Properties** — It is a colourless odourless crystalline solid with melting point 70–71° C. Soluble in alcohol and propylene glycol solutions being stable and unaffected by light heat dilute acids or alkalis. Solutions mix freely with glucose saline and barbiturates. Supplied in 10 ml ampoules of 10 per cent solution dissolved in equal volumes of propylene glycol and ethyl alcohol.

**Pharmacology** — Has a strong anti-strychnine and anti-tetanus effect probably acting on the basal ganglia and reducing the excitability of spinal cord reflexes. It relieves thalamic pain and by removing the muscular tenseness associated with emotional strain has some use as a sedative. It does not interfere with the passage of impulses across the myoneural junction consequently anticholinesterases do not antagonize its action. Action not influenced by adrenaline ephedrine or atropine nor do the usual anaesthetics speed recovery after its use. It is absorbed slowly from the alimentary canal and when suitably dissolved can be injected intramuscularly. It does not potentiate barbiturate anaesthesia. Excreted partly by the liver after conjugation with glycuronic acid most of it being outside the body within one hour of intravenous injection. Is not very good as a relaxant of the larynx. In its present form it is liable to cause thrombosis of the vein together with destruction of red cells haemoglobinuria and oliguria. Cases of death have been reported following its use presumably due to uraemia consequent on the blocking of renal tubules with products of red cell destruction.

More recent work tends to show that if the drug is given in strengths of 2 per cent or less red-cell destruction does not occur. It is the strength of solution which is important rather than the total amount injected.

When given to conscious patients nystagmus and defective convergence are the initial signs produced. Voluntary muscular power is not reduced. It has a deleterious effect in high concentration on the perfused heart. It has local analgesic properties.

*Mephenesin continued*

**Clinical Uses** —Is used more for the control of spastic states such as mild cases of tetanus strychnine poisoning status epilepticus than as a relaxant in anæsthesia where it now has few uses. Spasticity is a condition of hyperactive stretch reflex with after discharge together with a tendency for impulses to irradiate in the spinal cord. It is this hyperactivity which is inhibited by mephenesin so that the patient can use what voluntary power he has to the best advantage. It is stated to give relaxation of the muscles of the abdominal wall without the simultaneous production of respiratory depression but it is inferior to *d* tubocurarine chloride gallamine etc.

Average dose is up to 1 g (50 ml of 2 per cent solution) given shortly before the peritoneum is opened. It is irritating in its original strength (10 per cent solution) if it is injected into the tissues. Effect lasts twenty to thirty minutes and can be repeated. Can be given as an elixir 1 g in half an ounce or as tablets (0.5 g).

Because it depresses the thalamus it has been used in psychiatry for pharmacological leucotomy.

**BENZOQUINONIUM CHLORIDE (MYTOLON)**

This is a relaxing agent also known as Win-747. Its chemical composition is 2,5-bis(3-diethylaminopropylamino) benzoquinone bis benzyl chloride. It was synthesized in the U.S.A. in 1950 by Cavallito and co-workers. It is a red crystalline solid sparingly soluble in water. Used in 0.3 per cent solution (3 mg per ml). It is not antidoted by edrophonium or neostigmine although it obtains its effect by non-depolarization. It has anticholinesterase properties.

It is not very rapid in onset its maximal effects coming on in ten to fifteen minutes afterwards gradually passing off. It has no effect on the cardiovascular system gives no evidence of histamine release such as bronchoconstriction and does not affect transmission of autonomic impulses. It is a powerful vagotonic drug and causes salivation bradycardia and colic. Atropine antagonizes these effects. Respiratory depression does not last long. Is more useful if used with cyclopropane than with thiopentone. Is excreted in the urine which may be turned red by the drug.

Dose for intubation 4.5–9 mg for maintenance 4.5–6 mg every twenty to thirty minutes. With ether less is needed.

Not yet decided whether it is a depolarizer or a true curariform drug i.e. a competitive myoneural blocking agent. (See articles by Arrowood Julia *Anesthesiology* 1951 **12** 753 and Dundee J. W. Gray T. C. and Rees G. Jackson *Anæsthesia* 1952 **7** 134.)

**Excretion of Relaxants —**

1. Decamethonium gallamine and benzoquinonium are excreted by the kidney unchanged.
2. *D* tubocurarine and its dimethyl compound are partly excreted by the kidneys and partly metabolized.
3. Suxamethonium is completely metabolized.

## CHAPTER XIX

## REFRIGERATION ANALGESIA, ELECTRICAL ANÆSTHESIA

## REFRIGERATION ANALGESIA

Refrigeration analgesia is the application of cold to a localized part of the body to block local nerve conduction of painful impulses. It is not to be confused with induced hypothermia which is the application of cold in order to reduce the oxygen needs of the tissues during temporary interruption of the circulation.

A useful method of analgesia revived by Fredk M Allen of New York City\*. It involves chilling and not freezing of the tissues. It acts on all the cells of the part not just on the nerve cells as do other anæsthetics and analgesics. It was used by Baron Larrey during Napoleon's retreat from Moscow in 1812 and by James Arnott of Aberdeen in 1847. Benjamin Ward Richardson used an ether spray to produce refrigeration anæsthesia in 1867 while in 1938 Fay described cryotherapy for the relief of pain.

The effects of chilling —

- 1 Interference with conduction of nerve impulses
- 2 Reduction of metabolic rate and oxygen requirements
- 3 Inhibition of bacterial growth and infection
- 4 Retardation of healing

**Technique** — Before a tourniquet can be applied the tissue underlying it must be chilled for an hour by the close application of thin rubber ice bags. Any convenient site proximal to the operation site is chosen for the tourniquet which is then most carefully put on. A  $\frac{1}{2}$  in rubber tube is very suitable. If the limb below the tourniquet becomes congested the tourniquet is inefficient and must be reinforced. A pale blanched limb shows that the tourniquet is efficient.

A rubber sheet is placed beneath the limb and firmly fastened by a safety pin 2-3 in above the tourniquet. Cracked ice is now piled round the limb and kept in place by a few turns of a bandage round the rubber sheet. If the head of the bed is elevated the melted ice will drain into a bucket. The rubber sheet is undone occasionally to see that the limb is kept surrounded by ice. Patients do not complain of discomfort due to cold and seldom to discomfort due to the tourniquet. A small intravenous dose of morphine will control discomfort if it is present.

A loose tourniquet has the following disadvantages —

- 1 Complete refrigeration and analgesia are hindered

*Mephènesin continued*

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A loose tourniquet has the following disadvantages —

- 1 Complete refrigeration and analgesia are hindered

See Allen F M and Crossman, L. W., *Refrigeration Anæsthesia and Treatments* C. & R. Anand 1943 22, 5

*Technique continued*

2 Chilling of the remainder of the patient is favoured

3 Venous congestion in the limb increases amount of blood lost  
A tourniquet can be dispensed with —

1 When the limb is rendered ischæmic by arteriosclerosis trauma or embolism

2 When operation site is superficial e.g. skin grafts can be cut after the tight application of ice bags for two hours

Other methods of cooling involve bandaging round the limb thin rubber ice bags and immersing the limb in a bucket of ice and water if the operation is on the distal part of a limb. Electrical apparatus can also be used. With any of these methods the skin temperature should be about 5°C (40°F). If necessary a thermometer can be used to verify this.

Duration of refrigeration depends on the bulk of tissue. For a thigh 2–3 hours, for a leg or arm 1½–2 hours, for a hand or foot 1 hour, for fingers and toes 20–30 minutes. If the surgeon is prepared to inject local analgesic solution into major nerves as he comes to them, refrigeration time can be reduced by about 50 per cent. About three buckets of chipped ice are required for an amputation through the thigh.

The patient in his bed is brought to the operating theatre and when the surgical team is ready is placed on the table, the limb dried and painted and the operation proceeded with. Cold lotions are used and warm objects kept away from the wound. Analgesia will last about an hour if the tourniquet is kept efficiently in place.

When main vessels have been tied the tourniquet is released, spurting vessels are dealt with and the wound closed. No evidence of thrombosis is seen in the stump.

Analgesia lasts long enough after removal of the tourniquet for the wound to be sutured.

After dressing the stump can be surrounded by ice bags for two or three days if infection is present. In clean cases no further chilling is necessary as it delays healing.

Ulceration of tissue does not occur if undue pressure of ice bags and actual freezing are avoided.

The method can be used for the treatment of badly traumatized limbs requiring amputation.

Preliminary chilling reduces pain and infection and by limiting absorption of toxins lessens shock. The patient's general condition can be treated with blood plasma etc. and later amputation performed under refrigeration analgesia without producing further shock. In these cases the tourniquet is only applied an hour or two before operation.

**Advantages** — Specially indicated in amputation of leg in arterio-sclerotic or diabetic gangrene. Mortality strikingly reduced in these operations by this method of analgesia.

There is absence of post-operative pain, absence of shock, less chance of stump infection, less upset of the patient's general condition.

**Disadvantages.**—Fussy technique Delayed healing in arterio-sclerotic tissues.

Refrigeration analgesia can be used for operations on the fingers and toes hands and wrists etc Duration of chilling is 20-30 minutes and a tourniquet can be applied either at the base of a digit or above the wrist Healing in healthy wounds is not delayed Application of an ice-bag for 20-30 minutes over a superficial abscess or boil will enable incision to be performed painlessly

The pain from post-operative wounds can be eased if an ice-bag is placed over the wound which is protected by a double layer of cellophane kept in place by adhesive strapping

### ELECTRICAL ANAESTHESIA\*

First shown to be possible by d'Arsonval in 1890 high frequency currents were used Four types of electrical anaesthesia are described

(1) General (2) General and Spinal (3) Spinal (4) Local

Robinsonitch produced local analgesia using direct current interrupted 100 times per second

Spinal anaesthesia results if direct or interrupted direct current is applied between the lumbar spine and the neck

Leduc used an interrupted direct current to produce general and spinal anaesthesia in 1902 applying the current between the loin and the head

General anaesthesia results if an electric current is passed through the head after the fashion used in electro-narcosis

Electrodes have been applied to the head and a current of 135 milliamperes at 700 cycles per second at 15 volts has been used in humans It causes anaesthesia complicated by tachycardia and hypertension When similar currents were applied to dogs no histological changes were demonstrated post mortem †

W F Burge in the USA has studied electrical sleep As anaesthesia deepens the electro-negative potential of the conscious cortex decreases and finally becomes electro-positive Anaesthetics may produce unconsciousness by causing the cerebral cortex to give up negative charges thereby becoming electro-positive and so less active and responsive to reflexes

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\* See article by Lancelotti O *J ment Sci* 1949 85 1  
 † Knutson J C *Archs Ex Phys* and Krumm J *Ill An Sts Med* 1951 17 615



## CHAPTER XV

CHOICE OF ANÆSTHETIC, AS INFLUENCED  
BY TYPE OF OPERATION

## UROLOGY

No general anæsthetic should be given if uræmic coma is imminent. Ether has a toxic effect on diseased kidneys. Normal operative procedures cause no change in the renal circulation. Under anæsthesia the blood pressure is a poor guide to the renal circulation. During anæsthesia methamphetamine increases while noradrenaline and adrenaline decrease renal blood flow. Blood loss of up to 1500 ml does not cause renal damage unless it is prolonged and especially if associated with hypotension. So blood loss must be replaced if it is excessive.

Many patients undergoing urological operations are old, atheromatous, and suffering from diseased kidneys and cardiovascular system. A high blood urea may be associated with a decreased tolerance towards intravenous barbiturates which should be used especially carefully in cases which show this abnormality. For example moderate doses of thiopentone may cause very prolonged sleep in a uræmic patient anæsthetized by the agent alone e.g. for cystoscopy or suprapubic cystostomy. Similarly only small doses of thiobarbiturates should be used during prostatectomy if the patient is uræmic. Old patients are usually more placid and philosophical than young patients so are often good subject for regional analgesia. Premedication should be minimal and atropine used in all patients over the age of about 60 rather than scopolamine.

## 1 Nephrectomy —

**NERVE SUPPLY TO KIDNEY \***—The renal plexus arises by numerous small roots from the semilunar superior mesenteric and aortico renal sympathetic ganglia which in their turn derive supply from the three splanchnic nerves—predominantly from least splanchnic. The vagus probably takes no part in renal innervation. This bundle of nerve fibres is grouped into upper lower and posterior renal nerves which are in relationship to the renal artery. From the posterior renal nerve is given off the superior nerve to the ureter.

Lateral position throws strain on respiratory efficiency of patient. Use of kidney bridge increases this strain and further predisposes to hypoxia and post-operative atelectasis of contralateral lower zone.

Extradural or unilateral spinal analgesia with 12–14 ml of hypobaric nupercaine is useful when patient is fit and muscular. Owing to the discomfort of the lateral position light general anæsthesia

with cyclopropane is often a useful supplement. Analgesia must ascend to T 6. Intradural and extradural analgesia does not greatly influence the tone of the ureters.

Endotracheal gas-oxygen with a thiobarbiturate and a relaxant and perhaps pethidine makes a useful combination.

Rarely the pleura is damaged during kidney operations; the resulting collapse of the upper lung may in association with the handicapped lower lung prove dangerous unless controlled by manual inflation of the lungs via the reservoir bag preferably through an endotracheal tube.

*Transplantation of the ureter* requires maximal relaxation obtained either by extradural analgesia or by a muscle relaxant with light general anaesthesia. Intra and extradural analgesia by causing a tonic small and contracted colon has been known to interfere with the uretero-colic anastomosis.

## 2 Suprapubic Cystostomy—

**THE INNERVATION OF THE URINARY BLADDER**—The bladder, lower ends of the ureters and the prostate are supplied by filaments of the inferior hypogastric plexus. This is formed from (1) The sympathetic—ganglia T 12-L 3. (2) The sacral nerves—S 2, S 3 and S 4. Fibres from the sources pass first to the pre sacral or superior hypogastric plexus and from here fibres pass to the inferior hypogastric plexus. Stimulation of the sympathetic component causes contraction of the bladder neck and of the prostatic and seminal vesicular musculature and the ureteric orifices and the detrusor muscle.

In poor risk cases regional block (see p. 359). In average cases where relaxation is not required either thiopentone (with or without pethidine) with gas and oxygen, light cyclopropane or light ether. In stout patients the addition of a muscle relaxant may be necessary. Intra or extradural analgesia (up to T 10) is very seldom necessary.

**3 Suprapubic Prostatectomy**—Relaxation is required and considerable shock is produced. Extradural analgesia using 12 to 30 ml of 1.5 per cent lignocaine with or without light general anaesthesia gives good results especially if emphysema and bronchospasm are also present. Spinal analgesia to T 10 obtained by 1–1.500 nupercaine (10–12 ml), 1.4 to 1.6 ml of hyperbaric nupercaine or procaine 300–350 mg provides relaxation and lessens shock but like extradural analgesia it causes a fall in blood pressure which may embarrass the cardiovascular system and mask haemorrhage; bleeding may then occur when the patient is back in bed with a blood pressure recovered to near normal. The blood pressure can in these cases be maintained by suitable drugs e.g. a continuous intravenous noradrenaline drip. Unfortunately both adrenaline and noradrenaline by causing constriction of the renal vessels reduce the renal blood flow. Methedrine has the opposite effect. Blood pressure raising drugs should be given carefully to hypertensive patients and only after it is known that the block has acted successfully. This ensures against undue rise in pressure which might harm the inelastic vascular system.

**Urology—Suprapubic Prostatectomy continued**

A useful technique is that suggested by Jennings Marshall and described by Stanley Rowbotham\*. They point out that a spinal kept low such as 1 ml of hyperbaric nupercaine injected and followed by the tail-down position for ten minutes giving analgesia to S 1 will block the parasympathetic fibres from the second third and fourth sacral roots leaving the sympathetic fibres from the first and second lumbar roots an unopposed action (the same effect will follow sacral extradural block using 15–20 ml of 1.5 per cent lignocaine solution) vasoconstriction and contraction of the prostatic bed are thus produced. Analgesia and relaxation of the anterior abdominal wall must then be obtained by regional block (see p 359) or general anæsthesia with or without a muscle relaxant. Innervation of the vasa from T 12 and L 1.

Many workers prefer a light general anæsthetic e.g. thiopentone with gas-oxygen-trilene cyclopropane light ether (in the absence of diathermy). Relaxation can be produced or increased by a suitable muscle relaxant. Arrangements should be made to have intravenous dextrose or blood to hand in these cases.

**4 Transurethral Prostatectomy**—If general anæsthesia is used the explosion hazard must be remembered.

Thiopentone can be used if the operation is likely to be relatively short supplemented by gas and oxygen and perhaps trilene or pethidine.

Low spinal is very satisfactory and needs to be up to S 1. To render the dome of the bladder insensitive to distension block must be to T 10 and skin analgesia must reach the umbilicus. If in addition the vasa are to be tied a little local infiltration may be required in addition. This small operation demands a block up to T 12.

Suitable techniques are 1.2 ml of heavy nupercaine 1.5 ml of 5 per cent lignocaine with 3.1 per cent dextrose given in the sitting position (in each case diluted at least half and half with cerebrospinal fluid) 1 ml of 1 per cent amethocaine (10 mg) with 1 ml 10 per cent glucose in the third interspace given in sitting position metycaine 1 ml of 10 per cent solution with 1 ml of cerebrospinal fluid in third interspace procaine 75–100 mg with cerebrospinal fluid 1½–2 ml in third interspace. A good combination is amethocaine 8 mg with procaine 40 mg diluted to 3 ml with cerebrospinal fluid given in the second or third interspace.

Extradural lumbar or sacral block may be used for these cases while both it and low spinal block can be accompanied by minimal thiopentone if the patient is anxious.

Isotonic saline should be used for irrigation as ordinary water may cause hæmolysis and possibly such post-operative complications as lower nephron nephrosis and bronchospasm during operation.

**Cystoscopy**—Topical analgesia is fairly satisfactory e.g. xylocaine gel 2 per cent but should not be used after recent instrumentation or in the presence of bleeding from the urethra (*see p 369*) a 2 per cent solution of pyribenzamine (tripelennamine) the anti-histaminic drug can be used successfully in these patients as an analgesic. Cases with gross cystitis are often unsuitable for local analgesia as the distension of the bladder with irrigating fluid causes painful spasm.

If a general anaesthetic is used for cystoscopy the anaesthetist must provide (1) Complete loss of sensation (2) Relaxation of bladder sphincters and abdominal wall (3) Quiet breathing through a patent airway (4) Freedom from hazard of explosion. In the author's opinion this anaesthetic procedure can be difficult to accomplish smoothly e.g. in emphysematous old men with bronchitis. It should never be undertaken lightly and may require endotracheal intubation. Even in experienced hands anaesthesia for cystoscopy is often inelegantly given.

Thiopentone with gas and oxygen together with a relaxant is a suitable anaesthetic but prolonged recovery may interfere with pyelography. In patients with uræmia thiopentone dosage must be kept very low.

Extradural sacral block is very satisfactory but care must be taken to prevent analgesia from involving the renal pelvis if pyelography is contemplated and therefore it must not involve the eleventh and twelfth thoracic nerves.

Low spinal is also satisfactory e.g. nupercaine 0.5 per cent solution 1 ml in third interspace procaine 60–90 mg in 2 ml of cerebrospinal fluid given in sitting position.

**6 Circumcision**—In babies open ether. Ethyl chloride should be avoided induction being more safely carried out with V A M (vinesthene anaesthetic mixture) in children general anaesthesia. In adults thiopentone extradural sacral block or regional block (*see p 360*). Babies easily develop laryngeal spasm sometimes necessitating interruption of the operation. Adequate depth of anaesthesia before the infliction of surgical trauma will usually prevent what may be a very dangerous complication.

## DISEASES OF THE SUPRARENAL GLANDS

**Primary Adrenal Failure**—May be seen following

- 1 Removal i.e. adrenalectomy
- 2 Destruction e.g. Addison's disease adrenal apoplexy
- 3 Exhaustion e.g. starvation and toxæmia
- 4 Dysfunction virilizing hyperplasia

**Secondary Adrenal Failure (Primary Pituitary)**—May occur following—

- 1 Removal (hypophysectomy)
- 2 Destruction e.g. tumours Simmonds's disease
- 3 Inhibition—due to cortisone or corticotropin therapy

Death due to circulatory failure may occur in patients who having been on doses of cortisone for long periods are suddenly deprived

**Diseases of the Suprarenal Glands—Secondary Adrenal Failure continued**

of it before the stress of anæsthesia and operation. Such patients should receive at least two 100 mg injections of cortisone in the hours before operation and perhaps afterwards too and their blood pressure may require support from transfused fluid or pressor drugs. Thorn's test may be helpful. If 25 mg of ACTH is given intramuscularly a 50 per cent or greater fall in the eosinophil count following this is evidence of adequate adrenocortical response. A negative test shows that no cortical adrenal tissue is functioning.

- 1 Addison's Disease**—Patients with this complaint are susceptible to infection to loss of sodium chloride and to narcotics. These patients are likely to be debilitated, hypotensive and perhaps tuberculous. They are bad anæsthetic risks as Addisonian crises are easily precipitated. These start with loss of sodium chloride in the urine and so of large amounts of water. The circulating fluid is reduced while diarrhoea and vomiting make the plasma volume smaller. Dehydration and circulatory collapse follow and in addition there is often autonomic imbalance which interferes with their homeostasis. If such a patient is operated on and Addison's disease not diagnosed, severe post-operative collapse may occur. If the disease is however recognized, adequate pre-operative treatment greatly lessens the risk, although post-operative hypotension is likely. If it occurs it should be treated by blood plasma or the intravenous injection of cortisone. Pre-operative management consists in giving sodium chloride and fluid, dextrose and cortisone to maintain the circulating blood volume as near normal as possible. Neither hypotension nor anoxia must be produced by the anæsthetic technique. Thiopentone may cause a serious fall in blood pressure.
- 2 Suprarenal Apoplexy**—This is usually fatal and is often associated with meningococcal septicæmia. Hyperpyrexia and circulatory collapse are the usual modes of death. Rarely the condition comes to operation because of associated arterial embolism or peritonitis. Cortisone may be indicated.
- 3 Cushing's Syndrome**—This is often associated with or perhaps caused by cortical hypertrophy or tumour. Surgical removal may cause Addisonian crises and similar measures must be taken to combat them (*see above*). Pre-operative sodium chloride etc. should be given for two days pre-operatively and also post-operatively. Intravenous noradrenaline and cortisone may be required.
- 4 Pheochromocytoma\***—This is a tumour of the adrenal medullary cells of chromaffin origin which although histologically benign may be dangerous because of excessive secretion of adrenaline and noradrenaline. The growth may not be confined to the suprarenal but may occur wherever chromaffin tissue is found e.g. in the

paravertebral spaces near the great vessels of the abdomen in the organ of Zuckerkandl near the aortic bifurcation and in the celiac plexus. The patient exhibits hypertension either paroxysmal or continuous hyperhidrosis and elevated basal metabolic rate with some fever perhaps. The operation is to remove the tumour usually by the retropleural or retroperitoneal routes. The condition is diagnosed by clinical examination, certain radiological investigations and by pharmacological agents designed to raise the blood pressure e.g. histamine, methylcholine (mecholyl) and tetra-ethyl ammonium bromide or to lower it e.g. phentolamine (regitine) or piperoxan.

**Pre-operative care.** Perhaps intravenous dibenamine 5 mg/kg or other sympatholytic drugs to control blood pressure together with sodium chloride.

**Anæsthetic technique** must cater for: (1) Good relaxation—gallamine is not the best drug to use because it may cause tachycardia. (2) The possibility of accidental pneumothorax. (3) The effects of excess adrenaline or noradrenaline during operation when the tumour is manipulated. (4) Circulatory depression when tumour is removed. Hypoxia which stimulates medullary secretion must be avoided as also must cyclopropane. Excessive hypertension may be controlled by the intravenous injection of piperoxan hydrochloride 20 mg or phentolamine (regitine) 5 mg while a continuous intravenous drip of noradrenaline 4 mg per litre (which is not inactivated by phentolamine) or of phenylephrine 20 mg per litre may be needed during the first three post-operative days.

- 5 Bilateral Adrenalectomy.**—This may be done for carcinoma of the breast or prostate or malignant hypertension. Patients suffering from secondary carcinomatosis may require special care directed to their bones, their blood picture and their pleura—in case of effusion. Pre-operatively a salt free diet, diuretics, digitalis, sedatives. Cortisone 100 mg should be given one hour before operation and four times daily on the first three times daily on the second post-operative day and then in gradually decreasing amounts until it can be taken by mouth. Intravenous phenylephrine or noradrenaline during operation may be required and is likely to be needed in the post-operative few days. Later sodium chloride must be pushed. During operation thiopentone and pethidine are not usually needed in large amounts whereas doses of gallamine and *d* tubocurarine are apt to be greater than normal perhaps because of the deficient pseudocholinesterase in the blood of these usually ill patients. Extradural block often satisfactory.

## ABDOMINAL SURGERY

**Essentials of Good Technique.**—Good technique must provide —

a Safety

b Good relaxation of the muscles of the anterior abdominal wall and peritoneum. As the rectus and the transversus muscles are accessory muscles of respiration their tone persists in planes of third stage anaesthesia which produce complete flaccidity.

Abdominal Surgery—Essentials of Good Technique *continued*

of the muscles of the limbs. The posterior fascial sheath of these two muscles is fused with the peritoneum in the upper abdomen so they must be well relaxed when the peritoneum is sutured. Relaxation is aided by flexing the head and neck and by raising the knees 6 in. from the table.

- c Quiet breathing. With ether when intercostal paralysis is complete the diaphragm compensates by increased and sometimes jerky movement. This is not seen with cyclopropane which produces quieter breathing (although at a greater risk of causing respiratory acidosis). Most abdominal operations are now performed under some type of intermittent positive pressure respiration.
- d Protection from shock and circulatory depression.
- e Minimal disturbance of respiratory function after operation from chemical irritation of the mucosa from prolonged hypoventilation of the lungs or from prolonged depression of the cough reflex. Upper abdominal relaxation can only be produced coincidentally with intercostal paresis so that tidal exchange is always reduced when this relaxation is present unless breathing is assisted or controlled.
- f Minimal interference with body chemistry.
- g Protection of the airway from aspiration of gastric contents. The risk of such aspiration is not of course confined to abdominal operations. If it is suspected that vomitable material is present in the stomach the successful passage of a stomach tube will make certain and through it liquid can be aspirated. The Ryle tube has three markings on it corresponding to the average distance between the upper teeth and the pylorus, the fundus and the cardia. A more efficient tube is the semi stiff œsophageal tube (e.g. size 12/7 mm diameter) through which can be sucked a larger volume of fluid together with small solid particles.
- h Pleasant induction and relative freedom from post operative sequelæ.

*For producing relaxation of the anterior abdominal wall* subarachnoid block although it is not at present popular is the most efficient technique. Next comes extradural block followed closely by the use of a muscle relaxant. The contraction of the gut associated with the sympathetic paralysis is often advantageous whereas the lack of cardiovascular depression is one of the strongest points favouring the use of the muscle relaxants. Then follows the use of deep ether and some way after that cyclopropane. Thiopentone, halothane and vinesthene cannot be relied upon for this purpose while trilene and nitrous oxide-oxygen are quite inadequate if used alone.

**Agents**—The occasional anaesthetist will produce the best results with ether using either an open mask or a gas machine. The E.M.O. inhaler is very useful for maintaining ether anaesthesia. Ether is well tolerated by most patients while n of safety  
is wide be made muscular relaxation is good and

reasonably quiet. As it is a respiratory stimulant the blood carbon-dioxide level is not so likely to rise as is the case with some other anæsthetic agents. Gas and oxygen can be used for induction. The semi-closed or closed circuit may be used for maintenance. Unfortunately ether has many well known disadvantages.

*Cyclopropane* is most useful in experienced hands. It produces quiet breathing and less post-operative chest complications than ether. Relaxation cannot be guaranteed but can be produced by the addition of a muscle relaxant, minimal ether or regional analgesia. With slow deepening of anaesthesia in patients of moderate musculature cyclopropane is often adequate when used alone. Preliminary examination of the patient's abdominal wall will tell the experienced anaesthetist what agent or technique will be necessary to produce good relaxation.

A large endotracheal tube is of great help in many upper abdominal operations.

*Thiopentone* is only suitable for abdominal operations in thin asthenic patients or in operations of short duration. It should be accompanied by gas and plenty of oxygen. It is not a good agent for the production of abdominal relaxation but is excellent when combined with a relaxant. It can be used however to produce extra relaxation for a short time e.g. while the peritoneum is being sutured in patients under inhalation anaesthesia.

*Extradural lumbar block* is a neglected but in the author's opinion an excellent method of providing for the surgeon a patient who is completely relaxed, has contracted bowels and in whom afferent impulses are blocked. Lignocaine 1.5 per cent solution—with or without amethocaine hydrochloride—is used in doses of 20–45 ml. For prolonged operations an extradural catheter should be inserted and serial injections given.

*Spinal analgesia* finds its most useful field in abdominal operations. The higher the level of analgesia obtained the less safe does it become. Thus it is safer for lower abdominal section—to T 9 or 8—than for upper laparotomies T 5 or 4.

High spinal is useful in fit asthenic muscular individuals. The quiet breathing, complete relaxation and contracted bowels produce good operating conditions.

Mid spinal i.e. to T 9 or 8 is safe for patients who are reasonably fit.

It is usually preferable to combine intra- or extradural analgesia with light general anaesthesia either with thiopentone, gas and oxygen or cyclopropane. A safety factor is to have the patient breathe an oxygen rich atmosphere. A supply of pressor drugs and an open vein into which to inject them must always be to hand.

The *muscle relaxants* are most useful to produce relaxation. They can be combined with cyclopropane or thiopentone (with or without pethidine) and gas and oxygen.

Good results follow the combination of thiopentone induction (up to 0.5 g. of 2½ per cent solution), gas and oxygen (at least 25 per cent), intravenous injections of 10–25 mg. of pethidine.



**Abdominal Surgery—Agents continued**

as required and a muscle relaxant such as *d* tubocurarine or gallamine or a drip of suxamethonium. Patients so treated awaken soon after operation and do not require analgesic drugs for several hours. As pethidine takes some minutes to act it must be given well in advance of its need. The average patient requires about 1 mg per minute. Signs of too light a plane of anæsthesia are (1) Small movements of hands face head or hips and knees (2) Increasing resistance to assisted respiration (3) A rising pulse rate (4) Sweating of face

*Regional analgesia* in expert hands gives good results. It can take the form of intercostal block (see p 349) or abdominal field block (see p 355) combined with either posterior or anterior splanchnic block (see p 350). Light general anæsthesia can be combined with regional analgesia the latter producing the necessary relaxation.

**Perforated Peptic Ulcer**—The operation is usually short the patient is usually shocked he is very liable to post operative chest complications. As absorption of toxic material by the lymphatics of the diaphragm is directly proportional to the excursion of the latter a technique producing quiet breathing is beneficial when peritonitis is present. Cases with this condition are usually acute emergencies i.e. their stomachs are likely to contain vomitable material. Moreover the surgeon is likely to handle and compress the stomach expelling some of its contents into the œsophagus. For these reasons a cuffed endotracheal tube should usually be passed to protect the air passages from contamination. Precautions must be taken to prevent aspiration of gastric contents during induction of anæsthesia and these will in many cases include the passage of a Ryle or œsophageal tube. The experienced anæsthetist may care to pass the cuffed tube with the head of the table tipped upwards using either cyclopropane and oxygen in equal proportions or in stronger and less handi-capped patients thiopentone. In either case a relaxant will probably be used. Beginners might be wiser to use gas oxygen and ether with the table tilted downwards and with an efficient suction catheter near at hand in case of vomiting during induction. It must be remembered that a stomach tube can lie unresisted in the trachea so before such a tube is used for irrigation its accurate position must be verified. Intercostal block with splanchnic block is a more elegant but more difficult technique (See pp 349 352)

**Acute Intestinal Obstruction**—Factors to be considered are —

- 1 The degree of shock present
- 2 The presence or absence of regurgitation or vomiting. The former is a passive process requiring no muscular force the latter is a muscular reflex act. The former is aided by a head down tilt and rendered less likely if the head is tilted upwards
- 3 The degree of distension
- 4 The degree of electrolyte imbalance

In both normal people and those with intestinal obstruction much fluid is excreted by the proximal small intestine only to be reabsorbed lower down. In high obstruction this subsequent reabsorption is prevented hence vomiting and interference with fluid balance. Low obstruction gives rise to distension. Vomiting causes loss of chlorides and alkalosis and consequently great fluid loss and dehydration. Distension causes interference with circulation of the bowel wall and pressure on the great veins causing reduced venous return to the heart a low blood pressure and interference with cardiac action.

Biochemical changes in intestinal obstruction include (1) Hæmo concentration (2) Reduction of fixed base in serum (3) Diminution of plasma chlorides (4) Increased blood urea and non protein nitrogen (5) Increase in carbon dioxide combining power of plasma (6) Acid urine with perhaps ketone bodies and low urinary chloride.

The stomach should always be emptied by either a Ryle or a wider bore œsophageal tube preferably the latter. Either of these can be passed through the nose after preliminary cocaineization. It should be introduced in the conscious patient such introduction being made a little less unpleasant by sedative premedication together with instructions for the patient to suck an amethocaine lozenge beforehand. Grave illness does not make this any less necessary. It should remain in place during the anæsthesia and until the return of the reflexes. If this precaution is not taken the risk of aspiration bronchopneumonia is great not to mention the possibility of asphyxia from inhaled vomit. Once the patient loses consciousness and the œsophageal sphincter relaxes gastric contents if present tend to regurgitate. It may come up in large amounts during vomiting or as a small trickle which promptly unless prevented falls into the trachea unnoticed. A patient with increasing cyanosis tightly clenched jaws and faeculent material issuing from the nose is a truly terrifying sight and one which carries a bad prognosis. That which cannot be easily treated had better be prevented.

Intra and extradural analgesia produces good relaxation contracts the bowel and does not interfere with the cough reflex. It produces circulatory depression and is dangerous in shocked and debilitated patients e.g. where systolic blood pressure is below 100 mm Hg.

General anæsthesia is safer in all subjects. Suction applied to the stomach tube should prevent aspiration of vomitus. A cuffed endotracheal tube should be used in these patients. If regurgitation is very great endotracheal intubation should be performed under topical analgesia of the larynx followed by the rapid induction of general anæsthesia and preceded by gastric intubation and suction. Induction of anæsthesia with the patient in the reverse Trendelenburg position to prevent gravity regurgitation through a relaxed œsophageal sphincter and the use of a sucker are also factors which make for safety. The actual agents and techniques used to produce general anæsthesia vary with different workers. The following may be used. Cyclopropane

**Abdominal Surgery—Agents continued**

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urethral catheter must be passed and aspirated and maintained in the stomach until the return of the reflexes. Denis Browne recommends that a bootlace stitch should be placed in the peritoneum before the pyloric tumour is delivered from the wound. During the operation this can be drawn aside. It avoids the need for increasing greatly the anaesthetic depth during closure. Rectal thiopentone 2.5 per cent solution 15–20 mg/lb given in the operating room with the buttocks held together—preceded by an enema—is a good form of premedication. This can be followed by infiltration with 0.25 per cent solution of lignocaine (3.5 mg/lb) with adrenaline 1:400 000.\*

**Ventral Hernia**—General anaesthesia together with a muscle relaxant is suitable. Bucking and coughing on the endotracheal tube during extubation with consequent strain on suture lines can be prevented by the injection of 20–50 mg of suxamethonium and oxygen insufflation before the tube is withdrawn although there are pharmacological objections to giving depolarizing relaxants to patients who have had non-depolarizing drugs throughout the operation.

For fit adult patients extra or intradural analgesia can be used to facilitate wound closure.

**Operations for Portal Hypertension**†—The patient needs to be fixed to the table in such a position that the incision can if necessary extend far laterally on each side and so that venograms can be taken. The patient's arms are flexed and raised so that the hands rest on the opposite shoulder where they are supported. The position is similar to that adopted at the commencement of the hornpipe dance. The position may be useful in surgery of the biliary tract and the lower end of the oesophagus.

**Gastrectomy**—Before operation many surgeons like the patient to swallow a Ryle's tube so that the stomach can be aspirated and kept empty. Other surgeons favour the passage of an oesophageal tube after anaesthesia has been induced.

**PASSAGE OF RYLE'S TUBE IN CONSCIOUS PATIENT**—The patient sits up and a well lubricated tube (glycerin or liquid paraffin) is inserted into a patent naris. The patient sips water while the tube is advanced with each act of swallowing. It should be inserted until the third mark is at the naris. A tube which is too soft should be discarded as it must not collapse when subjected to the negative pressure of the sucker. Distance from incisor teeth to cardia averages 17 in (43 cm). A Ryle's tube may be in the trachea without either the patient or the anaesthetist being aware of it.

**PASSAGE OF RYLE'S TUBE IN UNCONSCIOUS PATIENT**—Steen recommends the following excellent method. A well lubricated Ryle's tube is inserted into a No. 6 Magill nasotracheal tube so that its tip is just within the lumen of the larger tube.

Black G. W. and Love S. H. S. *Anaesthesia* 1957 12 430 and Leatherdale R. A. L. *Lancet* 1958 1 932.

† Hunt, A. H. et al. *Lancet* 1956 1 881.

**Abdominal Surgery—Acute Intestinal Obstruction** *continued*

thiopentone with gas oxygen and trilene ether A muscle relaxant will almost certainly be necessary

Spinal analgesia will not reduce distension due to ileus associated with peritonitis

**Operations associated with Hæmorrhage**—Cyclopropane is very good for these cases Intradural and extradural block are seldom indicated Full oxygenation through a large patent airway is most important Blood or plasma volume expander in its absence must be transfused

**Abdominoperineal Resection of Rectum**—Maximal relaxation is necessary and contracted intestines an advantage Extra or intradural analgesia with light general anæsthesia or a muscle relaxant with light general anæsthesia are suitable As there is often considerable shock produced blood drips etc must be set up If the surgeon requires a steep Trendelenburg position the use of an endotracheal tube is desirable The technique advised by Ronald Jarman is as follows (a) Omnopon scopolamine premedication (b) Thiopentone 0.5 g with patient sitting (c) Light nupercaine 14–15 ml given in the third lumbar interspace the patient remaining sitting upright for 55–60 seconds (d) Patient placed supine in slight Trendelenburg position (e) Gas and oxygen given with more thiopentone if necessary (f) No drips given until patient is back in bed

The writer prefers—in the usual patient who is submitted to this operation (i.e. an oldish rather handicapped patient) either a combination of thiopentone pethidine relaxant and gas oxygen given through a large orotracheal tube or a continuous or single injection extradural block A blood drip is set up in a vein If an extra length of plastic tubing is used the arm can be secured to the table as it lies against the side of the patient Intravenous drugs can be given either into the tubing or more elegantly into a televenous apparatus (*see* Chapter XIII)

**Rectosigmoidectomy**—Heavy nupercaine 12 ml diluted with cerebrospinal fluid injected with patient's head elevated followed by the level supine position together with light general anæsthesia Patients do equally well with light general anæsthesia together with a relaxant

**Ramstedt's Operation**—Frequently performed under local infiltration of the abdominal wall using up to 15 ml of 0.5 per cent procaine and adrenaline During the twenty four hours preceding operation fluids must be pushed and the infant must come to the theatre with its stomach washings clean Premedication is neopenthe 1 min hypodermically or chloral hydrate 5 gr given 2 hours before operation The child is securely bandaged to a cross splint and during the operation is fed a mixture of honey and syrup of choral from a dummy The patient must be kept warm

Open ether preceded by V A M induction also gives good results but before administration of a general anæsthetic a 6 gauge

*Hiccup* is a troublesome reflex too often associated with present day methods of light anaesthesia. Its exact cause is ill understood but it is likely to be associated with carbon dioxide build up and light anaesthesia. The best method of prevention is the use of adequate amounts of a relaxant drug. It may also yield to (1) Inhalation of amyl nitrite (2) Inhalation of concentrated ether vapour for a few breaths (3) Inhalation of ammonia (4) Intravenous injection of methedrine (5) Increased depth of general anaesthesia (6) Block of vagus nerves near cardia. This also has prophylactic value.

**Operations on the Biliary Tract**—Calcium and vitamin K analogue should be given before operation. Anaesthetic technique is similar to that used for gastrectomy. There is in prepared patients not the same need to use a cuff on the endotracheal tube. The dynamics of the biliary ducts must be considered in connexion with pre and post-operation pain relief in patients with biliary disease. Normally the increase in intrabiliary pressure required to overcome the tone of the sphincter of Oddi does not cause pain. Analgesics (e.g. morphine and pethidine) may by stimulating this sphincter to contract increase the intrabiliary pressure up to 20 cm. of water and thus produce pain. Parasympathetic stimulators have a similar effect. To relieve biliary colic therefore the dose of analgesic must be great enough to cause cerebral depression. The following drugs lower intrabiliary pressure by relaxing the sphincter: (1) Amyl nitrite (2) Nitroglycerin gr 1/50 (3) Papaverine 2 gr. Atropine even up to gr 1 is disappointing in this respect.

Cases of acute haemorrhagic pancreatitis are bad anaesthetic risks no matter what agent and method are used so that obscure abdominal emergencies should have serum amylase tests done so that operation can be avoided.

**Sigmoidoscopy**—If general anaesthesia is required for this successful administration may not be without difficulty. The patient is usually required to lie on his side. A suitable technique is thiopentone a relaxant and gas-oxygen. Dilatation of the anal sphincter may cause laryngeal spasm or other respiratory difficulty unless the reflex mechanism is subdued and in certain cases endotracheal intubation may be desirable. Extradural sacral block is satisfactory in these cases too.

## NEUROSURGERY

Local analgesia with 0.5-1.0 per cent procaine 0.5 per cent lignocaine or 1-0.000 amethocaine or nupercaine is often satisfactory for short operations (see p. 321). Adrenaline should be added so that the final strength of solution is 1-100,000. Adrenaline saline of same strength is often used to reduce oozing even when general anaesthesia is employed. Periosteum is not sensitive and bone only slightly so, thus drilling though uncomfortable is usually tolerable. The dura is sensitive near the base of the skull and near the middle meningeal vessels but insensitive elsewhere. The cerebral cortex (except the post-central sensory

**Abdominal Surgery—Gastrectomy continued**

near the bevelled end. This is then passed blindly into the œsophagus from the nose being guided by the fingers or the laryngoscope if necessary. The Ryle's tube is now inserted well into the stomach through the Magill tube. When aspiration of stomach contents shows its correct position the Magill tube is withdrawn and the Ryle's tube held firmly against the posterior wall of the pharynx as the larger tube leaves the nose. Intermittent suction is kept up during the operation.

The author generally uses one of the following techniques. A (1) The intravenous injection of 0.3 to 0.5 g. of thiopentone in 2½ per cent solution through the televenous apparatus (see Chapter XIII) or through a Mitchell non clotting needle. (2) Through the same needle injection of suxamethonium 25–50 mg. of active cation (perhaps preceded by gallamine 20 mg. in order to avoid the muscular fasciculation of suxamethonium). (3) Inflation with mixture of nitrous oxide 6 l. and oxygen 2 l. (4) Spraying of cords with 4 per cent solution of lignocaine via a Macintosh laryngeal spray and a laryngoscope. (5) Insertion under direct laryngoscopic vision of a large (size 8–12) Magill rubber endotracheal tube with inflatable cuff. (6) Maintenance with additional doses of muscle relaxant and pethidine together with the gas and oxygen mixture in a closed circuit. B (1) Induction with thiopentone 200–250 mg. (2) Injection of gallamine 120–160 mg. or *d* tubocurarine 20–30 mg. (3) Inflation of the lungs with gas and oxygen. (4) Intubation with a large cuffed tube followed by controlled respiration with gas and oxygen and such additional doses of relaxant as may be necessary to keep the patient still relaxed and free from reflexes. (5) Neostigmine and atropine to restore normal ventilation.

The passage of a cuffed endotracheal tube is advisable as the surgeon may expel blood clot or other material from the stomach up into the œsophagus and pharynx even in the presence of a freely working Ryle's tube.

Alternative techniques are —

- 1 Cyclopropane muscle relaxant intubation
- 2 Thiopentone gas oxygen ether
- 3 Extra or intradural analgesia with light general anæsthesia
- 4 Intercostal block splanchnic block light general anæsthesia

The depth of anæsthesia needs to be greatest —

- 1 During the incision of the skin
- 2 During the initial abdominal exploration
- 3 While the cardiac end of the stomach is under tension
- 4 During the closure of the peritoneum

The aim should be to have the patient breathing deeply moving about the bed and coughing as soon after the end of the operation as possible.

**The Celiac Plexus Reflex** — Mechanical stimulation of the celiac plexus (by retractors packs the gall bladder bridge etc.) may result in sudden reflex arterial hypotension. This autonomic reflex may be obtunded by . . . (Irvine)

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*Neurosurgery continued*

cortex) is insensitive to pain stimuli. Muscle must be infiltrated if seen. Local analgesia is unsuitable for encephalography owing to the pain following air injection. *Diminution of blood flow in cerebral arteries* is associated with (1) A fall in general blood pressure (2) Reduction of carbon dioxide tension in blood as by overventilation (3) Inhalation of high oxygen mixtures (4) Elevation of head and neck. *Increase in cerebral blood flow*—undesirable in neurosurgery—is associated with (1) A rise in systemic blood pressure (2) Carbon dioxide excess. Inhalation of 7 per cent carbon dioxide increases the cerebral blood flow 75 per cent (3) Oxygen lack (4) Respiratory obstruction (5) Coughing. Cerebral vessels are supplied by sympathetic vasoconstrictor nerves.

Heroin gr  $\frac{1}{2}$  given by mouth eases the pain of cerebral tumour without producing much increase of intracranial pressure. Phenacetin also is a good drug.

Pethidine and alphaprodine (nisentil) given intravenously both raise the intracranial pressure. This effect is reversed in the case of pethidine by nalorphine and in the case of alphaprodine by levallorphan. The other effects of the drugs are not significantly reversed.

When general anaesthesia is used in most cases an endotracheal tube should be passed. It should be well lubricated with an analgesic to help prevent coughing. It must be securely attached to the patient so that it with its breathing tubes is not pulled out during the operation.

It must be of the largest size consistent with an atraumatic introduction—usually size 10 or 12 Magill and should be unlikely to become kinked. The Portex tube is better than rubber in this connexion while the newer rubber tubes embodying a wire coil are better still. Oral intubation is usually preferable to nasal as thereby a larger tube can be employed. It must not be too long for fear of its distal end irritating the sensitive carina causing cough. Its distal end should be cut at right angles and not bevelled so that the close application of a bevel to the tracheal wall with consequent obstruction to free gas flow is avoided. Bad anaesthesia may prevent a successful operation being performed.

Bucking following extubation causes a rise in cerebrospinal fluid pressure. It can be prevented by injecting 20–50 mg suxamethonium and inflating with oxygen before the tube is removed. The cerebrospinal fluid pressure can easily rise due to faults on the part of the anaesthetist. A smooth induction and intubation are very important. Hypoventilation must not be tolerated. A non rebreathing technique given adequate tidal exchange is probably better as a means of avoiding hypercapnia than a closed circuit. Ether given carefully probably does not cause much rise in pressure.

The technique must —

1. Keep the patient quiet and free from coughing and straining during both induction and maintenance. Cough increases bleeding by raising the intracranial venous

- 2 Provide the lightest plane of anaesthesia ensuring the above
  - 3 Minimize venous oozing by avoiding ether and avoiding respiratory obstruction hypoxia and build up of carbon dioxide by hypoventilation. In long operations the sucker may be necessary to ensure the patency of the tube
  - A poor airway promotes oozing in three ways —
    - a The resulting hypoxia is accompanied by increase in intracellular fluid
    - b The resulting hypercapnia produces venous dilatation
    - c The increased expiratory effort causes a rise in intrathoracic pressure and so increases venous pressure and oozing
  - 4 Avoid agents which are toxic when used for long periods
  - 5 Avoid the risk of explosion when the diathermy is used
- Ether causes a rise in intracranial pressure while the barbiturates produce a fall

In operations done in the sitting position injury to veins may be followed by air embolus. In this position hypotension may be caused by quite reasonable doses of thiopentone or pethidine

A high or rising pulse rate may call for cerebral ventricular puncture either before induction or during maintenance of anaesthesia

The following techniques are suitable —

- 1 Thiopentone induction followed by gas oxygen and intermittent pethidine or minimal trilene using a semiclosed technique or Ayre's T piece
- 2 As above utilizing halothane or chloroform in minimal dosage rather than trilene. The combination of adrenaline with both chloroform and trilene is theoretically dangerous
- 3 Intermittent thiopentone with gas and plenty of oxygen. Topical laryngeal analgesia and the use of a muscle relaxant facilitate intubation. This method is not suitable for the longer operations but can be used for example in prefrontal leucotomies or exploratory trephines
- 4 Open ether given on a mask placed above the endotracheal tube. Ether-air is not as explosive as ether-oxygen and the method can be made relatively safe by arrangement of towels
- 5 Gas-oxygen and minimal ether in a closed circuit
- 6 The use of controlled respiration has some advantages. It reduces intracranial pressure and should include a negative phase \*

The use of a muscle relaxant will facilitate direct vision endotracheal intubation

A semiclosed non rebreathing circuit by keeping the tension of rebreathed carbon dioxide to a minimum gives good results. Either a No 10 Portex tube or a No 10 Magill rubber tube protected from kinking by a Bourne's (flexible) metal tube can be used. The breathing tubing should be carried to the machine across the patient's chin and not over his forehead. Careful watch should be kept on the blood pressure and pulse rate. Morphine should be avoided in cases with raised intracranial pressure for fear of producing respiratory depression. The

**Neurosurgery continued**

patient should be very light at the end of the operation so that the surgeon can be assured that any prolonged unconsciousness is not due to anæsthetic drugs

Hypotensive techniques have been used successfully during intracranial operations especially those for blood vessel tumours

Hypothermia has a definite place in neurosurgery and allows important vessels to be temporarily occluded during the removal of vascular tumours \*

**Posture during Operations on the Spine ††**—Bleeding from the extradural veins is the chief trouble. Pressure on the chest or abdomen coughing and straining squeeze blood out of the abdominal and thoracic veins into the vertebral veins so distending them. The ideal position (if the surgeon cannot be persuaded to operate with the patient on his side) is the jack knife position with the prone patient's pelvis supporting his weight. This allows the extradural veins to collapse. The field of operation should be the highest point on the operating table. A head down position is contra indicated if a myelogram has been performed. The use of a cuffed endotracheal tube gives control of the airway and prevents aspiration of stomach contents. A muscle relaxant by reducing intra abdominal pressure will reduce bleeding. Adrenaline 1-250 000 in saline can be infiltrated into the skin and muscles.

For laminectomy in the lumbar region a light general anæsthetic given through an endotracheal tube is usually employed. Otherwise an extradural spinal analgesic can be used. The patient may be semi prone or fully prone in the so called Mohammedan praying position. If a general anæsthetic is given and the operation performed with the patient prone a stomach tube should be passed to avoid aspiration of stomach contents into lungs as regurgitation may follow pressure on the patient's lumbar region. In these cases a well padded rest should be placed beneath each shoulder and beneath the pelvis so that a small interval separates the patient's chest from the table. This will aid respiration and reduce bleeding by preventing an increase in the abdominal and hence in the extradural venous network. Also it will prevent circulatory depression resulting from pressure on the inferior vena cava. (A R Hunter)

To maintain blood pressure during neurosurgical operations neosynephrine 10 mg (1 ml) added to 1 l of 5 per cent glucose intravenously is useful. In all neurosurgical operations likely to last some time an intravenous drip should be set up and arrangements made to check the blood pressure at regular intervals.

To reduce intracranial pressure the intravenous injection of 100 ml of 50 per cent sucrose may be used.

Lucas, B G B *Brit med Bull* 1958 14 46

†Taylor A R, Gleadhill, C A, Bilsland W L, and Murray P F *Brit J Anaesth* 1956 28 213

‡Pearce D J *Proc R Soc Med* 1957 50 107

**Anæsthetic Management of Hypophysectomy**—See articles by  
Ballantine R I W *Anæsthesia* 1956 11 303 Tung I I  
Randall H T and Howland W S *†Anesthesiology* 1956  
17 739

**Cerebral Angiography**—This was popularized by Egas Moniz of Portugal—of leucotomy fame—in 1927 and developed by Engeset in 1944 and by Lindgren in 1947. It is usually done through the intact skin when 40 per cent diodone is injected into a carotid artery average dose about 30 ml. Local or general anæsthesia can be employed and if the latter an endotracheal tube should be passed using e.g. thiopentone and a relaxant. Coughing and movement must be avoided during the examination and during intubation so as to avoid the production of congested veins in the neck. Swelling of the neck from extravascular contrast medium or from effused blood may cause respiratory obstruction.

**Lumbar encephalography**—In children and uncooperative adults an endotracheal tube of non kinkable type is inserted and through it gas oxygen and trilene are given. Attention must be given to the airway as the radiologist will require the neck to be acutely flexed.

#### **Ventriculography**—

- 1 In children endotracheal general anæsthesia
- 2 In adults local analgesia

**Management of Head Injuries**—These patients should be assumed to have vomitable material in the stomach. A stomach tube should be passed in an attempt to empty the stomach while as good an airway as possible must be ensured by posture, an artificial airway, control of the jaw and tongue or an endotracheal tube. After twenty four hours of intubation a tracheotomy should be performed if the airway is still obstructed. Any narcotic or anæsthetic drug may depress respiration if the intracranial pressure is high. Routine tracheotomy has been recommended for all severe head injuries to prevent pulmonary complications.

If aspiration of foreign material into the bronchi has already taken place e.g. blood associated with the injury or stomach contents then bronchoscopy may be a life saving measure.

(See also articles by Hunter A R and Hewer A J H *Proc R Soc Med* 1952 45 427)

### **CLEFT PALATE AND HARE LIP IN INFANTS\***

The repair of cleft palate is usually done in two stages while the hare lip can be repaired during one of these operations or done independently. Many surgeons prefer to operate when the child is about 10–16 weeks old for hare lip and about one year old for the palate. The child should be fit and healthy.

Atropine  $\frac{1}{4}$ – $\frac{1}{2}$  gr is suitable as premedication and for children over the age of 2 pentobarbitone 0.6 gr per stone.

\* See also articles by Davies R M and Danks S J *†J Dent* 1953 11 275 Norman W D and Lees R C O *Br J J Dent* 1955 27 527

**Neurosurgery continued**

patient should be very light at the end of the operation so that the surgeon can be assured that any prolonged unconsciousness is not due to anæsthetic drugs

Hypotensive techniques have been used successfully during intracranial operations especially those for blood vessel tumours

Hypothermia has a definite place in neurosurgery and allows important vessels to be temporarily occluded during the removal of vascular tumours \*

**Posture during Operations on the Spine ††**—Bleeding from the extradural veins is the chief trouble. Pressure on the chest or abdomen coughing and straining squeeze blood out of the abdominal and thoracic veins into the vertebral veins so distending them. The ideal position (if the surgeon cannot be persuaded to operate with the patient on his side) is the jack knife position with the prone patient's pelvis supporting his weight. This allows the extradural veins to collapse. The field of operation should be the highest point on the operating table. A head down position is contra indicated if a myelogram has been performed. The use of a cuffed endotracheal tube gives control of the airway and prevents aspiration of stomach contents. A muscle relaxant by reducing intra abdominal pressure will reduce bleeding. Adrenaline 1-250 000 in saline can be infiltrated into the skin and muscles.

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Atropine  $\frac{1}{2}$ — $\frac{1}{4}$  gr. is suitable as premedication and for children over the age of 2 pentobarbitone 0.6 gr. per stone.

\* See also articles by Ballantine R I W and Lindgren W S *Anæsthesia* 1956 11 303 and Tun, P P Randall H T and Howland W S *Anesthesiology* 1956 27 739

*Cleft Palate and Hare lip in Infants continued*

Endotracheal anæsthesia is indicated using a Magill cleft palate tube (sizes 00 0 1 and 2) (Fig 58). This consists of rubber distally and a coiled spring covered by thin rubber proximally where there is also a small gas side delivery tube. It can be used as an Ayre's T piece by fixing to the proximal end 4-6 in. of rubber tubing to allow a little re-breathing. The Magill tube is kept rigid during oral introduction by a metal stylet. The portex tube is also useful for these cases. The oral route should be used when possible as it allows the passage of a larger tube. Care is necessary to see that kinking of the tube does not occur nor must it be occluded by the gag. The patient's hands should be firmly secured. Anæsthesia can be induced on an open mask with vinyl ether or ethyl ether or a nitrous oxide cyclopropane

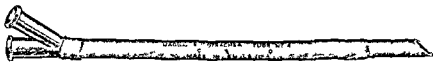


Fig 58—Magill flexo intubation tube (British Oxygen Co. Ltd.)

ether oxygen sequence can be used. Other workers give gas oxygen and ether through an Ayre's T piece while there are those who use thiopentone (2 to 5 mg/lb) pethidine (2-7 mg/stone) and *d* tubocurarine (1 mg/5 lb) or gallamine (1 mg/lb). If a rapidly acting relaxant is used for intubation the dose (of suxamethonium) should be 0.5 mg/lb intravenously or 1 mg/lb intramuscularly with hyaluronidase or 2 mg/lb intramuscularly without hyaluronidase. Reflex resistance to intubation must be abolished. Intubation is facilitated if the glottis is first moistened with cocaine solution from the tip of a finger. Introduction of the endotracheal tube may be hindered by an anteriorly displaced premaxilla obstructing the field of vision. To prevent the blade of the laryngoscope from sinking deeply into the wide cleft gauze packing or adhesive tape can be used or the gap can be bridged by a spatula. Dott's gag is useful as it allows the endotracheal tube to lie in the longitudinal slot in its blade. At least 30 per cent of oxygen should be given throughout and volatile or non volatile agents may be used to supplement the gas and oxygen. A total flow of 2-3 l a minute is adequate once the patient has settled down the gases flowing over the surface of the volatile anæsthetic. Only light anæsthesia is required.

Following removal of the tube and a careful oropharyngeal toilet a patent airway can be ensured by a suture through the tongue. Post operative œdema of glottis should be prevented by gentle technique and adequate depth of anæsthesia during intubation. Treatment is steam tent oxygen tent or tracheotomy. With gentleness cleanliness and care it should rarely be seen.

**HEMORRHOIDECTOMY**

The choice is between —

- 1 Light general anaesthesia with a muscle relaxant
- 2 Extradural sacral block—15–20 ml of 1.5 per cent xylocaine or 1½ per cent metycaine (see p 372)
- 3 Thiopentone (0.5 to 1 g) and a relaxant (e.g. flaxedil 40–100 mg) gas and oxygen should be given at the same time. Occasionally the complication of laryngeal spasm will require in addition to the use of a muscle relaxant cyclopropane or ether or trilene. If this troublesome accident shows itself it may well be prudent to ask the surgeon to interrupt his highly stimulating manipulations until control of breathing has once again been obtained.
- 4 Low spinal (S 4–5) e.g. procaine 60 mg or nupercaine 0.5 per cent 0.6 ml diluted with cerebrospinal fluid between L 4 and L 5 with patient sitting. In men of prostate age spinal analgesia is better avoided so that it will not be blamed for any disorders of micturition which may arise post-operatively.
- 5 Local infiltration with procaine. From a point 1 in posterior to the anus with the index finger of the left hand in the rectum 2 per cent procaine-adrenaline solution is injected total amount 50 ml. Only one site of injection used and anus and anal canal are ensheathed by a cylinder of procaine solution.

Proctocaine which is frequently used after anal operations contains butyl-*p* aminobenzoate (butesin) 6 per cent benzyl alcohol 5 per cent procaine base 1.5 per cent in sterile almond oil. It should be warmed before use to make it less viscous and should not be injected immediately beneath the mucosa. Eufocaine can be similarly employed. Tubadil long acting  $\delta$  tubocurarine is said to relieve the pain after removal of piles. It causes reduction of sphincter tone the effect coming on 30 minutes after intramuscular injection and lasting for twelve hours. The dose is 1 ml twelve hourly.

**FACIO MAXILLARY OPERATIONS**

In anaesthetizing these very difficult cases the following points must be emphasized. (1) Airway must be ensured and maintained before operation if there is obstruction by a traction stitch through the tongue or by the tonsil position. The patient if he is unconscious may have already inhaled blood or vomit before arriving in hospital. (2) Because of possible obstruction to breathing no sedative premedication should be given. (3) The upper air passages should be sucked out before induction by bronchoscope if necessary. (4) Induction may be by thiopentone with a muscle relaxant or by intravenous bromethol in 1 per cent solution. Cyclopropane may also be used. (5) Naso-tracheal intubation under direct vision preceded by spraying of the cords with local analgesic solution allows the surgeon to have access to the mouth and pharynx etc. (6) The pharynx should be packed off after intubation. (7) Careful tracheobronchial suction must be performed after operation. (8) The patient must be returned to the ward in the tonsil position (semi prone) and attention must be directed to his airway until his reflexes return.



**Facio-maxillary Operations continued**

Before completion of any jaw fixation a careful tracheo-bronchial toilet must be carried out and pack must be removed. A light plane of anæsthesia is desirable at the termination of the operation. Wire cutters should be handy in case of respiratory obstruction due to inhalation of blood or vomit.

If severe obstruction is present preliminary tracheotomy under local analgesia is advisable if the anæsthetist doubts his ability to intubate. Glottic œdema without actual obstruction is no bar to intubation.

As in all acute accident cases the possibility of a full stomach must be borne in mind and if thought necessary an œsophageal tube should be passed before anæsthesia is induced. See also excellent article by Thornton H. L. and Rowbotham S. *Anæsthesiology* 1945 6 380.

**EAR, NOSE, AND THROAT SURGERY\***

In these operations the following factors should be considered —

- 1 Premedication must be adequate but not heavy enough to cause a sluggish cough reflex after operation.
- 2 Induction must be smooth to reduce the incidence of bleeding.
- 3 A large tube should be used to avoid even minor degrees of respiratory obstruction.
- 4 Tidal exchange must be adequate to prevent hypercapnia and hypoxia.
- 5 No topical analgesic must be applied to the larynx or trachea as the cough reflex must be brisk after operation.
- 6 Entrance of blood and pus into the chest must be prevented by the use of an inflatable cuff on an endotracheal tube and/or efficient pharyngeal packing.
- 7 The operation should be done with the patient in a slight reverse Trendelenburg position to minimize venous oozing.

Induction with thiopentone intubation under a relaxant and maintenance with gas oxygen and trilene with or without pethidine can usually be relied on to provide a safe and smooth technique.

**SKIN GRAFTS**

Skin grafts can often be removed painlessly from the thigh after block of the external femoral cutaneous and if necessary the anterior crural nerves or by intradermal and subcutaneous infiltration of skin of anterior part of thigh. Work has also been done on superficial freezing of donor areas by ice bags.

**INTRANASAL OPERATIONS**

If topical or regional analgesia is not used general anæsthesia should be maintained through an orotracheal tube sealed off with a cuff or a pharyngeal or nasopharyngeal pack. If the last method is chosen a small right angled retractor is used to lift the soft palate forward by its free edge. The post nasal space can then be firmly packed with marine sponges on tapes or with moist gauze. These packs should not

be removed until the return of the patient's cough reflex. Forgetfulness to remove a pharyngeal pack on the other hand is one of the easiest mistakes for an anaesthetist to make and it can readily prove fatal. Should orotracheal intubation prove difficult nasotracheal intubation is carried out blindly and the nasal end of the tube brought out through the widely opened mouth after its retrograde withdrawal from the nose. Muscle relaxants will aid intubation but care must be taken to see that the cough reflex is active at the end of the operation.

### REMOVAL OF TONSILS

The best basal narcotic is rectal paraldehyde because of its slight depressant effect on the cough reflex but rectal thiopentone is very satisfactory if the nursing supervision before and after operation is skilled and alert. Methypentynol has been recommended \* 500 mg or two drachms for ages 2-4 750 mg or three drachms ages 4-8 and 1 g or four drachms ages 8-10 one hour before induction of anaesthesia. Nephenthe 1 minim per year is injected when consciousness returns. Hyoscine gr  $\frac{1}{8}$ - $\frac{1}{4}$  can be given by mouth with the sedative before operation.

For the guillotine operation nitrous oxide-oxygen induction open or closed ethyl chloride vinesthene or trilene followed if necessary by a little ether. It is necessary to produce relaxation of jaw muscles—the masseters pterygoids and temporales to allow easy insertion and opening of the gag relaxation of the palatoglossus in the anterior pillar and of the palatopharyngeus in the posterior pillar and of the superior constrictor of the pharynx the pharyngeal reflex must be absent but coughing must return quickly.

Premedication is atropine alone. The teeth are examined and any loose ones noted if these are disturbed they must be accounted for. A Doyen gag is inserted and if possible opened while the child is conscious and he is then anaesthetized preferably commencing with gas and continuing with open ethyl chloride. Children over five may require in addition a little ether. The tonsils are removed in the supine position with the child breathing air and he is then immediately turned on to his side while the adenoids are curetted. He returns to his bed in the tonsil position a welter of blood sweat and tears.

For dissection an endotracheal tube is preferred by many objected to by some. It can be passed either through the nose or through the mouth.

**Technique of Intubation in Children**—Ethyl chloride is given until about ten stertorous breaths have been taken then a lubricated Portex tube of suitable curvature (obtained by boiling it on a curved stylet) is passed blindly from the nose into the trachea. Ethyl chloride can be preceded by gas and oxygen. For children between 3 and 5 years of age size 2 is suitable over 5 size 3. Larger children taking of course a larger tube. If the patient regains his reflexes before the tube finds the glottis more

**Removal of Tonsils—Technique of Intubation continued**

ethyl chloride can be given and a further attempt made at intubation. The deep breathing and relative insensitivity of the larynx produced by ethyl chloride make it a very suitable agent for intubation in children. Only rarely after some experience will it be necessary to deepen anaesthesia with ether and use the laryngoscope. Some workers prefer to intubate under thiopentone and a relaxant or gas oxygen and a volatile agent.

The use of a tube allows the plane of anaesthesia to be more easily controlled, reduces oozing and obviates respiratory obstruction. The alternative is endopharyngeal insufflation in which case the patient should be taken well down into Plane 3 before the mouth is opened and the operation commenced. If this is not done it may be difficult to maintain adequate anaesthesia until the end of the operation.

Gas oxygen ether, gas oxygen trilene with or without thiopentone and a relaxant can be used. The aim should be to have the patient coughing within a minute or two of the completion of the operation.

Adenoids can be curetted either with the endotracheal tube in situ or after its withdrawal when the cough reflex has returned.

The patient should be returned to the ward in the tonsil position (semi prone, prevented from rolling on to his face by a pillow beneath the chest and prevented from rolling supine by bringing the lower arm behind the body) and remain in this position until full consciousness is regained.

Anaesthesia for the bleeding tonsil can be a grave responsibility. The patient is likely to have a stomach full of blood clot and to be shocked. After atropine premedication and with the patient on his side in the Trendelenburg position anaesthesia is perhaps most safely induced with gas oxygen trilene and ether. The patient is then turned on his back, his pharynx is sucked clear of blood and an oral endotracheal tube is passed. A nasal tube after adenoid curettage might not be welcomed by the surgeon. Post operative suction and blood transfusion are both likely to be necessary.

**FENESTRATIONS**—The anaesthetist may well be required to take active steps to reduce bleeding. The following techniques have been recommended—

- 1 The use of trimetaphan or hexamethonium
- 2 The use of chloroform or halothane
- 3 The use of the phenothiazine drugs \* The present author \* prefers the last method

**TOTAL LARYNGECTOMY**

The available methods are—

- 1 With no pre-existing tracheostomy. A cuffed endotracheal tube is passed and a tracheostomy performed towards the end of the operation.

- 2 Tracheostomy performed immediately before the operation under general or local analgesia—the insertion of a cuffed tube through the opening
- 3 Anaesthesia through a pre existing tracheostomy opening \*  
For laryngofissure a cuffed tube is inserted from the mouth while the early dissection is done. After a planned tracheostomy a smaller (number 5 or 6) cuffed sterile short tube is placed in the trachea by the surgeon and connected to the anaesthetic machine

A good description of the technique employed can be consulted in *Anaesthesia* (1950 5 21) in an article by Chester and Lewis. After omnopon and scopolamine premedication they spray the upper air passages and larynx thoroughly with cocaine from a Rowbotham's spray and after injecting 0.5 to 0.75 g of thiopentone pass a large cuffed Magill tube through a laryngoscope into the trachea. The use of a relaxant makes this easier. Maintenance is by gas-oxygen and serial thiopentone injections. Inflation of the cuff protects the lungs from blood etc. As the stages of the operation proceed the proximal end of the tube is cut and is delivered into the neck. Later the freed larynx is threaded off the tube and a short cuffed number 5 or 6 tube inserted directly into the trachea by the surgeon the tube being reconnected in each case to the gas machine via a sterile angle piece. Deflation of the cuff takes place at the conclusion of the operation after careful aspiration by sucker.

### BRONCHO ŒSOPHAGOSCOPY

The special dangers of bronchoscopy are (1) Toxic effects from local analgesic drugs: it is unwise to exceed 5 ml of 4 per cent cocaine, 5 ml of 4 per cent lignocaine or 9.75 ml of 1 per cent amethocaine (with 0.25 ml of 1:1000 adrenaline). (2) Asphyxia due to blood, pus or mucus e.g. when a biopsy is performed. In wet cases local analgesia is often safer than general anaesthesia. Such patients should be sent back to bed in the tonsil position to aid drainage. (3) Reduced ventilation during general anaesthesia or under general or local anaesthesia when the bronchoscope is far down one main bronchus. (4) Bronchial or laryngeal spasm: this likelihood makes the use of topical analgesia necessary even if general anaesthesia is used in addition. (5) Trauma due to straining and movement of the patient.

The best method of anaesthesia for bronchoscopy and examination of the larynx is in the author's opinion that described by Macintosh †. The patient after suitable premedication is given intravenous injections through a Mitchell needle of thiopentone—say 300 mg and suxamethonium 80 mg. After inflation with oxygen the larynx is carefully sprayed with a solution of local analgesic and the pyriform fossae are similarly painted. With a long spray some solution either 4 per cent cocaine or 4 per cent lignocaine is carried down to the carina. The respiration soon returns and the patient breathing spontaneously is controlled by intermittent doses of thiopentone.

Coffin, S. *Anaesthesia* 1955 10 259.  
† Macintosh, R. R., *Ibid* 1954 9 77.

## Broncho œsophagoscopy continued

- 1 TOPICAL ANALGESIA —See section in chapter on REGIONAL ANALGESIA (p 368) Topical analgesia is safer than general anæsthesia if the patient has respiratory obstruction. It enables in addition the surgeon to assess the movements of the vocal cords. This cannot be accurately done if the patient is anæsthetized.
- 2 GENERAL ANÆSTHESIA —Patient premedicated with either a barbiturate and atropine or with omnopon-scopolamine. Patient placed on table with a Mitchell needle in a suitable vein. A muscle relaxant e.g. flaxedil is injected intravenously starting with 20 mg and followed if no untoward results are seen by another 20 to 60 mg. Then thiopentone 2½ per cent solution is injected amount about 0.4–0.5 g. After adequate inflation of oxygen under pressure the patient is ready for bronchoscopy and oxygen is given down the side tube of the bronchoscope during the operation. If active breathing stops the end of the bronchoscope can be occluded by the finger while oxygen is insufflated into the lung down the side tube. A rapidly acting relaxant can also be used successfully instead of flaxedil such as suxamethonium chloride 30–50 mg repeated if required. Prolonged apnoea in cases so treated may be associated with (1) Overdose of suxamethonium (2) Overdose of thiopentone (treatment—oxygen) (3) Hyperventilation with oxygen or (4) A low plasma cholinesterase level (treatment—fresh blood transfusion).

Before œsophagoscopy an endotracheal tube should be passed through the nose or mouth into the trachea and gas and oxygen and trilene inhaled during the examination. For successful œsophagoscopy obtunded reflexes, a good airway and a relaxed cricopharyngeus muscle are necessary.

These techniques are smoothed by preliminary topical analgesia. Intravenous pethidine alone—50 to 100 mg injected slowly without any premedication—or topical analgesia has given good results for œsophagoscopy and gastroscopy.\*

Insufflation of 3 litres a minute of oxygen down a small plastic tube placed in the trachea while the patient is under the influence of thiopentone and suxamethonium will keep the patient pink during bronchoscopy and unless apnoea is prolonged will not result in very harmful hypercapnia.

If massive hæmorrhage occurs from the pulmonary artery during biopsy either a bronchus blocker should be passed into the main bronchus which is bleeding or an endobronchial tube should be passed into the uninjured side.

For account of a technique dispensing with endotracheal intubation see article by Goligher J C and Thornton H I *Lancet* 1957 **I** 652.

IN CHILDREN deep ether followed by air and trilene down the side tube of the bronchoscope. A muscle relaxant is often helpful e.g. d-tubocurarine chloride 2–3 mg per stone.

of body weight. Basal narcosis produced by bromethol or rectal thiopentone or by rectal paraldehyde is most useful in children before bronchoscopy and together with topical local analgesia may be all that is required. Gas oxygen and chloroform is sometimes used for bronchoscopy in children. It combines deep anaesthesia with absence of risk of explosion.

### BRONCHOGRAPHY

Lipiodol was introduced as an antisiphilitic by Lafay in 1901 and used as a contrast medium in 1922 by Sicard and Forestier. The water soluble contrast medium is more irritant than lipiodol. Instillation by needle through the cricothyroid membrane has lost favour. Peroral instillation using a head light, laryngeal mirror and laryngeal syringe may result in some of the oil being swallowed.

**IN CHILDREN**—Techniques employing the instillation of contrast medium through an endotracheal tube either directly or through a rubber catheter are satisfactory in adults but in children are likely to cause partial asphyxia.

The following methods have been recommended—

- 1 It has been pointed out that when the swallowing reflex has been abolished by general anaesthesia iodized oil (or any other material e.g. blood vomit) placed in the pharynx will be sucked into the bronchial tree on inspiration.\* Premedication is restricted to atropine and general anaesthesia is induced by thiopentone ethylchloride vinesthene gas and oxygen etc and maintained by open drop ether carried to the plane where the swallowing and coughing reflexes are obtunded (stage 3 plane 3). A few breaths of carbon dioxide are given the mouth is opened and 20 ml. of contrast medium is poured into the pharynx. After a few deep breaths the oil is aspirated into the bronchial tree and radiographs can be taken. Immediately afterwards the patient is inverted and placed prone and remains so until the return of the cough reflex has resulted in most of the oil being expectorated. Post radiological suction is rarely necessary but pre radiological postural drainage is essential † (See letter by Temple L. J. and Gray T. C. *Brit med J* 1952 2 15).
- 2 Endotracheal intubation of a small Magill 00 catheter or length of polythene tubing through which the warmed oil is injected the child breathing through its natural air passages (Macintosh and Mushin *Brit med J* 1950 1 1319).
- 3 Rectal bromethol (100 mg/kg) and atropine premedication. Larynx sprayed either before or after a little gas oxygen and trilene or open trilene. Urethral catheter or polythene tubing (internal diameter 1.7 mm) inserted between the vocal cords for  $\frac{1}{4}$  in. The fine tubing can be made rigid using piano wire. A small volume of 2.5 per cent cocaine solution is injected down the tubing into the trachea and the tubing emptied of solution. When coughing has subsided the

\* Jacobs A. M. and Heats, G. *Lancet* 1935 2, 191.  
† Littlehouse J., *Brit. J. Anaesth.* 1953 29 407.

## Broncho œsophagoscopy continued

- 1 TOPICAL ANALGESIA—See section in chapter on REGIONAL ANALGESIA (p 368) Topical analgesia is safer than general anæsthesia if the patient has respiratory obstruction. It enables in addition the surgeon to assess the movements of the vocal cords. This cannot be accurately done if the patient is anæsthetized.
- 2 GENERAL ANÆSTHESIA—Patient premedicated with either a barbiturate and atropine or with omnopon-scopolamine. Patient placed on table with a Mitchell needle in a suitable vein. A muscle relaxant e.g. flaxedil is injected intravenously starting with 20 mg and followed if no untoward results are seen by another 20 to 60 mg. Then thiopentone 2½ per cent solution is injected amount about 0.4–0.5 g. After adequate insufflation of oxygen under pressure the patient is ready for bronchoscopy and oxygen is given down the side tube of the broncho scope during the operation. If active breathing stops the end of the bronchoscope can be occluded by the finger while oxygen is insufflated into the lungs down the side tube. A rapidly acting relaxant can also be used successfully instead of flaxedil such as suxamethonium chloride 30–50 mg repeated if required. Prolonged apnoea in cases so treated may be associated with (1) Overdose of suxamethonium (2) Overdose of thiopentone (treatment—oxygen) (3) Hyperventilation with oxygen or (4) A low plasma cholinesterase level (treatment—fresh blood transfusion).

Before œsophagoscopy an endotracheal tube should be passed through the nose or mouth into the trachea and gas and oxygen and trilene inhaled during the examination. For successful œsophagoscopy obtunded reflexes, a good airway and a relaxed cricopharyngeus muscle are necessary.

These techniques are smoothed by preliminary topical analgesia. Intravenous pethidine alone—50 to 100 mg injected slowly without any premedication—or topical analgesia has given good results for œsophagoscopy and gastroscopy.\*

Insufflation of 3 litres a minute of oxygen down a small plastic tube placed in the trachea while the patient is under the influence of thiopentone and suxamethonium will keep the patient pink during bronchoscopy and unless apnoea is prolonged will not result in very harmful hypercapnia.

If massive hæmorrhage occurs from the pulmonary artery during biopsy either a bronchus blocker should be passed into the main bronchus which is bleeding or an endobronchial tube should be passed into the uninjured side.

For account of a technique dispensing with endotracheal intubation see article by Goligher J C and Thornton H L *Lancet* 1951 **1** 652.

IN CHILDREN deep ether followed by air and trilene down the side tube of the bronchoscope. A muscle relaxant is often helpful e.g. d-tubocurarine chloride 2–3 mg per stone.

of body weight. Basal narcosis produced by bromethol or rectal thiopentone or by rectal paraldehyde is most useful in children before bronchoscopy and together with topical local analgesia may be all that is required. Gas oxygen and chloroform is sometimes used for bronchoscopy in children. It combines deep anaesthesia with absence of risk of explosion.

### BRONCHOGRAPHY

Lipiodol was introduced as an antisiphilitic by Lafay in 1901 and used as a contrast medium in 1922 by Sicard and Forestier. The water soluble contrast medium is more irritant than lipiodol. Instillation by needle through the cricothyroid membrane has lost favour. Peroral instillation using a head light laryngeal mirror and laryngeal syringe may result in some of the oil being swallowed.

**IN CHILDREN**—Techniques employing the instillation of contrast medium through an endotracheal tube either directly or through a rubber catheter are satisfactory in adults but in children are likely to cause partial asphyxia.

The following methods have been recommended—

- 1 It has been pointed out that when the swallowing reflex has been abolished by general anaesthesia iodized oil (or any other material e.g. blood vomit) placed in the pharynx will be sucked into the bronchial tree on inspiration\*. Premedication is restricted to atropine and general anaesthesia is induced by thiopentone ethylchloride vinesthene gas and oxygen etc. and maintained by open drop ether carried to the plane where the swallowing and coughing reflexes are obtunded (stage 3 plane 3). A few breaths of carbon dioxide are given the mouth is opened and 20 ml. of contrast medium is poured into the pharynx. After a few deep breaths the oil is aspirated into the bronchial tree and radiographs can be taken. Immediately afterwards the patient is inverted and placed prone and remains so until the return of the cough reflex has resulted in most of the oil being expectorated. Post radiological suction is rarely necessary but pre radiological postural drainage is essential† (See letter by Temple L. J. and Gray T. C. *Brit med J* 1952 2: 15).
- 2 Endotracheal intubation of a small Magill 00 catheter or length of polythene tubing through which the warmed oil is injected the child breathing through its natural air passages (Macintosh and Mushin *Brit med J* 1950 1: 1319).
- 3 Rectal bromethol (100 mg/kg) and atropine premedication. Larynx sprayed either before or after a little gas oxygen and trilene or open trilene. Urethral catheter or polythene tubing (internal diameter 1.7 mm) inserted between the vocal cords for  $\frac{1}{2}$  in. The fine tubing can be made rigid using piano wire. A small volume of 2-3 per cent cocaine solution is injected down the tubing into the trachea and the tubing emptied of solution. When coughing has subsided the

Jacoby N. M. and Keat G. *Lancet* 1938 2: 191.  
 † Parkhouse J. *Brit J Anaesth* 1955 29: 447.



Bronchography in Children *continued*

child is positioned and the contrast medium injected into the lungs—this will take several minutes. The dose of cocaine safe for a 10-stone adult is 100 mg so a safe dose for a child can be worked out according to its weight (Mushin W W and Lake R *Anæsthesia* 1951 6 88)

- 4 General anæsthesia followed by endotracheal intubation of part of a small cuffed tube which has been cut through above the cuff leaving the side tube available for injection of the warmed iodized oil. Anæsthesia is maintained by gas oxygen and trilene via an Ayre's T piece (M H A Davison)
- 5 Endotracheal intubation without general anæsthesia (See letter in *Brit med J* 1950 1, 24 by E G Sita Lumsden) The co-operation of the child is sought atropine is given and is followed by a dose of linctus codem and an amethocaine lozenge to suck. The patient sits while his larynx is sprayed with 2 per cent butyn. Then a Jacques catheter No 5 or 6 English (No 9 can be used for adults) is lubricated with analgesic jelly and is passed by the nasal route into the hypopharynx and 0.5 to 1.5 ml of butyn is injected into its proximal end. When coughing has disappeared the tube is manipulated into the larynx and secured in place by strapping. The patient can be positioned as required and oil can be instilled down the tube. Success is claimed even in six year old children
- 6 Yet another method is to pass a bronchoscope under thio pentone suxamethonium and perform careful suction after inflation with oxygen. This is followed by careful inspection the bronchoscope is replaced by an oral endotracheal tube with suction union and instillation of iodized oil radiography suction. Small doses of a muscle relaxant added to thio pentone gas-oxygen trilene will produce the general anæsthesia. Ether vapour may be ignited by the small bulb of the bronchoscope which gets very hot

IN ADULTS—Premedication atropine and 500 mg of methyl pentynol. Patient sitting up putting out his tongue with his chin forwards and slightly upwards. Number 9 Jacques catheter smeared with 2 per cent lignocaine ointment inserted over the tongue blindly into the trachea. Coughing soon settles down. Contrast medium injected down catheter and patient positioned. (Barker J C *Brit med J* 1955 1 1931) An amethocaine lozenge sucked and a local analgesic solution sprayed into the larynx may be used in addition

## ORTHOPÆDIC OPERATIONS

Manipulations requiring good relaxation for a short time are conveniently done under thiopentone a relatively large dose being given just before the surgeon is ready to produce his trauma. A relaxant can be added if required

Children who may require several operations e.g. talipes should be well premedicated

Leg amputations may be done under unilateral spinal analgesia a method well tolerated in old people undergoing amputation for gangrene care must be taken to keep the block unilateral by maintaining the lateral position for 20-30 minutes after sub arachnoid injection Otherwise thiopentone gas-oxygen cyclopropane or refrigeration analgesia can be used

Brachial plexus block is most useful for operation on the upper limb including reduction of shoulder dislocations (p 333)

For insertion of the Smith Petersen type pin unilateral spinal analgesia up to T 12 gives good relaxation e.g. nupercaine 0.5 per cent 1.4 ml or procaine 100-120 mg The patient usually tolerates being turned on to his injured side quite well The operation can be done under local infiltration analgesia with careful premedication Amethocaine 1-2000 is infiltrated from the skin to the bone in the line of incision An attempt is made to deposit solution between the fractured bone-ends from above the great trochanter and also from a point just external to the femoral artery located by its pulsation

Thiopentone with gas and oxygen and if necessary minimal trilene is also a satisfactory technique Ventilation is often made easier if an endotracheal tube is passed In elderly patients amount of thiopentone should be kept as small as possible

### THYROID OPERATIONS

**Pre-operative Preparation**—The patient must be made by the physician safe for surgery Rest in bed full diet sedatives iodine digitalis and thiouracil may be necessary The last agent will sometimes produce a basal metabolic rate lower than normal in the thyrotoxic patient by inhibiting the production of thyroid hormone and if this is so pre operative sedation and basal narcosis must be prescribed with the fact of hypometabolism in mind With modern preparation the need for multiple stage operations is much reduced It is important to check the possibility of respiratory obstruction from retrosternal goitre compression of the trachea or its deviation

Bad signs are (1) Failure of pulse rate to become less than 100 (2) Auricular fibrillation this does not contra indicate operation (3) History of previous heart failure (4) Failure to gain weight under medical treatment (5) Prolonged existence of disease (6) Vomiting diarrhoea

**Premedication**—Omnopon  $\frac{1}{2}$  gr and scopolamine  $\frac{1}{8}$  gr depending on metabolic level and general condition of patient Atropine because it raises the basal metabolic rate and increases the heart rate is better avoided

Stealing the thyroid is now seldom necessary and the use of bromethol is not as valuable as it formerly was If used the dosage should be 100 mg per kilo or 110 mg per kilo in very toxic cases Omnopon and scopolamine should be given in half dosage in addition Langton Hewer recommends rectal paraldehyde (1 drachm per stone of body weight with 1 oz as a maximum dose) for his thyrotoxic patients while rectal thiopentone is also a good basal narcotic

**Thyroid Operations** *continued*

**Anæsthetic Agents**—All the commonly used agents have their advocates Gas and oxygen gas-oxygen and ether gas-oxygen and trilene gas-oxygen and thiopentone with or without intra venous pethidine Some use cyclopropane routinely others fear it in the presence of cardiovascular abnormality Intermittent doses of thiopentone or pethidine can be injected into the ankle veins during the operation if the patient is not settling well

**Airway**—Some are in favour of endotracheal airways some are against them Endotracheal airways avoid respiratory obstruction and hypoxia with ooing without their use post operative tracheitis and mucus collection is lessened and a lighter plane of anæsthesia is tolerated

A tube is almost essential (1) If on examination of the radiograph the trachea is deviated or compressed (2) If goitre is retro sternal (3) If malignancy is suspected (4) If the vocal cords are functioning abnormally when viewed through a laryngeal mirror in husky stridulous patients (5) In recurrent cases

If in doubt it is better to intubate before the operation than during its course The author uses a tube routinely

Where pre operative stridor is present intubation should be performed under topical laryngeal analgesia preceded by a period of inhalation of 100 per cent oxygen

Stimulation of the recurrent laryngeal nerve causes spasm of the corresponding cord with a high pitched crowing sound when patient is not intubated If nerve is divided the cord first becomes abducted and flaccid later it assumes the cadaveric position between abduction and adduction Later still some voluntary control is gained

**Techniques of Intubation**—(1) Intravenous injections of a muscle relaxant (e.g. suxamethonium 30–100 mg) and thiopentone followed by direct vision oral intubation This should not be attempted in anatomically difficult patients (2) After 0.2 to 0.4 g of 2½ per cent thiopentone solution the nose pharynx and larynx are efficiently sprayed with a topical analgesic e.g. 4 per cent cocaine The tube is passed into the nasopharynx and gas and oxygen and if desired a little trilene are given Then tube is slipped into trachea Failure can be followed by re application of mask and a further attempt

**Conduct of Anæsthesia**—The eyes should be oiled and protected from the mask towels etc

Cuffed tubes should not be used The author prefers a semi-closed circuit with a non rebreathing valve The arms and legs of the patient should be firmly secured to prevent inconvenient movement during the operation at the light plane of anæsthesia necessary During the operation the blood pressure and pulse rate should be charted The curve of the latter is more important than the actual rate which increases on intubation while the incision is being made and during mobilization of the gland If all is well the rate decreases towards the end

of operation. The trachea and pharynx should be aspirated with a sucker at the conclusion of the operation. If 20-50 mg of suxamethonium are given before extubation and the patient then inflated with oxygen the cords can be examined at the time when respiration returns to see that they move normally.

The carotid sinus syndrome is occasionally seen (Heymans and others 1933). Irritation of a carotid sinus produces sudden effects which may be (1) Vagal cardio-inhibitory with bradycardia—atropine being the remedy. (2) Vasodepressive the remedy being a pressor drug. (3) Cerebral. The operation should be temporarily stopped, the head of the table lowered, artificial respiration instituted and 10 ml of 1 per cent procaine injected near the bifurcation of the common carotid artery of the side being operated on. The intravenous injection of atropine 1-2 mg may help while good results have followed the intravenous injection of curare (Burstein).

Recently the author has been using the phenothiazine drugs with satisfactory results. After routine premedication the patient is placed on the operating table with a head up tilt. Chlorpromazine, promethazine and pethidine in a dosage of about 50 mg of each is injected intravenously preferably slowly via a drip. A small dose of thiopentone or hexobarbitone is now given preceded by gallamine 20 mg to prevent muscular fasciculation which might be caused by suxamethonium which is then given after an interval in a dose of 50-100 mg. The patient is oxygenated by intermittent pressure on the reservoir bag of the anaesthetic machine and his trachea, cords and pharynx are sprayed thoroughly with 4 per cent lignocaine. A large tube is then inserted well lubricated with xylocaine paste. Inflation is continued, a face piece carefully strapped in place and soon spontaneous respiration takes place of a mixture of nitrous-oxide-oxygen (75:25) with a flow rate of 8 litres a minute using a non return semi closed circuit (see p 163).

The technique reduces bleeding and attention is focused on

- (1) The head up tilt
- (2) Intubation without any straining
- (3) Large patent airway
- (4) Adequate respiratory minute volume
- (5) Absence of carbon dioxide rebreathing

**Regional Analgesia**—Regional analgesia may include bilateral deep and superficial cervical plexus block (see p 332) together with intradermal and subcutaneous infiltration of the line of incision. Even if general anaesthesia is to be used some surgeons like infiltration of the line of incision both deep and superficial to the platysma with adrenaline saline (adrenaline 1:1000 1 ml to 250-500 ml saline) to avoid wound oozing.

Deep anaesthesia is not required but the possibility of respiratory obstruction and the degree of toxicity of the patient must be borne in mind.

E. S. Rowbotham prefers to solve the problem as follows. Intradermal wheals are made (1) In the suprasternal notch (2) Just above the midpoint of the clavicles (farther out if the

**Thyroid Operations—Regional Analgesia** *continued*

goitre is very large) (3) On each side of the neck opposite the greater cornua of the hyoid bones. From each of these five points deep and superficial infiltration is carried out the superficial injections resulting in a layer of solution covering the whole area. Injections are made throughout with a moving needle and after negative aspiration tests before the deep injections. These given through the same wheals are designed to deposit solution—(a) on each side of the trachea (b) laterally to the goitre (c) around the superior poles (d) near the ansa hypoglossi. Using the upper cervical wheal the needle is advanced 2 in parallel to but beneath the anterior border of the sternomastoid beneath which lies the carotid sheath the ansa being anterior to the sheath—5 ml of solution is injected on each side as the needles are withdrawn. Rowbotham uses no more than 150 ml of 1-4000 amethocaine with 1-400 000 adrenaline solution.

**Respiratory Obstruction after Thyroidectomy**—The causes may be —

- 1 **ŒDEMA OF THE LARYNX** which is usually seen on the second or third day after operation. Diagnosis is by indirect laryngoscopy and if stridor becomes troublesome a tracheostomy will be required the tube being removed about five days later.
- 2 **RECURRENT LARYNGEAL NERVE INJURY**—This is most likely to occur after operations for recurrent thyroidectomy. It may be temporary due to bruising or permanent due to nerve division—the former may last up to six weeks. Paralysis of one recurrent nerve may or may not cause obstruction. Paralysis of both cords will make a tracheotomy necessary. Even slight obstruction may prove fatal in handicapped patients e.g. cardiac cases and tracheotomy is always better done early than late. Its delay is the most frequent cause of death after thyroidectomy.
- 3 **COLLAPSE OF TRACHEA**—This is rare unless the actual tracheal cartilage is removed in malignant cases. When this condition is suspected the following conditions must be first excluded: œdema of larynx, recurrent laryngeal nerve injury or hæmorrhage under the strap muscles.
- 4 **INJURY TO THE SUPERIOR LARYNGEAL NERVE**—This is rare but can be suspected if there is (a) A change in the voice (b) Difficulty in swallowing. The former is due to cricothyroid paraly is the latter to sensory paralysis. The condition soon improves.

Obstruction to respiration causing insomnia requires either tracheotomy or intubation.

**Post operative Care**—Fowler's position morphine when required rectal saline with 20-30 minims of Lugol's iodine aspirin for the muscular pain in the neck. The patient should be kept cool. Thyroid crises characterized by delirium restlessness and tachycardia are best treated in an oxygen tent.

# GYNÆCOLOGY

Abdominal operations often do well with spinal extradural analgesia combined with minimal thiopentone and gas-oxygen. Light nupercaine 10-12 ml or heavy nupercaine 1.4-1.7 ml are very satisfactory if spinal (intradural) analgesia is contemplated. The abdominal pack to cover the intestines should be inserted gently to avoid initiating reflexes from the upper abdomen. If general inhalation anaesthesia is used breathing must be quiet and relaxation good. The combination of a muscle relaxant with light general anaesthesia is very satisfactory but it does not produce the contracted bowels seen after intradural and extradural analgesia. Endotracheal intubation should be reserved for those patients thought likely to develop respiratory difficulty e.g. those with a short fat neck or an underdeveloped lower jaw. Obese hypertensive patients do not always tolerate an extreme Trendelenburg position without some hypoxia. They are improved if oxygen is given during the operation and the breathing is assisted or controlled.

The Trendelenburg position is unphysiological and should be maintained for as short a time as possible. The less steep it is the better for the patient's respiratory and cardiovascular function. Levelling of the table should be gradual.

Vaginal operations can be performed under extradural lumbar or sacral block (see p. 307) spinal analgesia, general inhalation anaesthesia or thiopentone given intravenously for short procedures. Stretching of the cervix or trauma to the perineum may produce laryngeal spasm requiring a deeper plane of anaesthesia or a small dose of a muscle relaxant. The author prefers lumbar extradural block for vaginal repair operations. It combines afferent block, muscular relaxation and moderate hypotension and ischaemia. The dosage of 1.5 per cent lignocaine solution used is 20-40 ml. Light thiopentone sleep can be employed if thought desirable. Otherwise heavy premedication may suffice. For dilatation and curettage thiopentone, gas and oxygen or gas-oxygen-trilene is excellent.

Autonomic reflexes arising in the pelvis may be (a) Pelvicardiac causing bradycardia and hypotension (b) Pelvilaryngeal causing adduction of the cords.

These reflexes can often be broken by curare (Burstein).

# EYE SURGERY\*

In intra-ocular surgery a sudden rise in intra-ocular tension following coughing, vomiting or straining may cause a severe disturbance and so in the past these operations have been usually performed under local analgesia. More recently cataract operations have been done under careful general anaesthesia taking great pains to avoid coughing and similar disturbances. Local analgesia has its disadvantages too. Old patients may be unco-operative while retro-ocular haematoma following injection of the local analgesic solution may, by increasing intra-ocular tension, seriously interfere with the success of the operation and may call for its postponement. General anaesthesia however

**Thyroid Operations—Regional Analgesia** *continued*

goitre is very large) (3) On each side of the neck opposite the greater cornua of the hyoid bones. From each of these five points deep and superficial infiltration is carried out the superficial injections resulting in a layer of solution covering the whole area. Injections are made throughout with a moving needle and after negative aspiration tests before the deep injections. These given through the same wheals are designed to deposit solution—(a) on each side of the trachea (b) laterally to the goitre (c) around the superior poles (d) near the ansa hypoglossi. Using the upper cervical wheal the needle is advanced 2 in parallel to but beneath the anterior border of the sternomastoid beneath which lies the carotid sheath the ansa being anterior to the sheath—5 ml of solution is injected on each side as the needles are withdrawn. Rowbotham uses no more than 150 ml of 1-4000 amethocaine with 1-400 000 adrenaline solution.

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- 3 **COLLAPSE OF TRACHEA**—This is rare unless the actual tracheal cartilage is removed in malignant cases. When this condition is suspected the following conditions must be first excluded: oedema of larynx, recurrent laryngeal nerve injury or hæmorrhage under the strap muscles.
- 4 **INJURY TO THE SUPERIOR LARYNGEAL NERVE**—This is rare but can be suspected if there is (a) A change in the voice (b) Difficulty in swallowing. The former is due to cricothyroid paralysis the latter to sensory paralysis. The condition soon improves.

Obstruction to respiration causing insomnia requires either tracheotomy or intubation.

**Post operative Care**—Fowler's position, morphine when required, rectal saline with 20-30 minims of Lugol's iodine, aspirin for the muscular pain in the neck. The patient should be kept cool. Thyroid crises characterized by delirium, restlessness and tachycardia are best treated in an oxygen tent.

superior alveolar branch of the second division of the fifth nerve (a) A wheal is raised 5 mm above the inner canthus at the orbital margin and through this solution is injected just within the orbit to block the infratrochlear nerve (See p 322—*anterior ethmoid block*) (b) The needle is partially withdrawn and solution is injected subcutaneously over the lacrimal sac avoiding the angular vein (c) The anterior superior alveolar nerve is blocked from a point 10 mm below the inner canthus (d) From the first wheal over the inner canthus a deep zone of infiltration is made to give hemostasis (e) The naris on the bad side is sprayed with cocaine solution and then packed with cocaine gauze or painted with cocaine paste (p 323) For excision of a chronically infected lacrimal sac infratrochlear nerve block and infiltration of the line of incision are all that is required

1 or dacryocystorhinostomy

- 1 The nasociliary nerve is blocked within the orbit (*anterior ethmoidal block* p 322)
- 2 The line of incision is infiltrated and this extends into the zone of skin supplied by the infra-orbital nerve

The operation can also be performed under general anaesthesia using adrenaline-saline infiltration or ganglionic blocking agents to provide ischaemia

**Retro ocular Block.**—This must be done before operation under local analgesia on the globe of the eye After topical analgesia of the cornea and conjunctival sac the long and short ciliary nerves are blocked within the muscle cone Retro-ocular block reduces intra-ocular pressure and makes prolapse of the vitreous less likely Hyaluronidase (6-10 turbidity reducing (T R) units to each ml of solution) aids diffusion and enables rather larger volumes of solution to be injected—up to 3 ml The injection should also paralyse the extra-ocular muscles

Retro ocular or retrobulbar block may be

- 1 Superior \* through the superior rectus with the patient looking downwards from a wheal just above the middle of the tarsal plate A 5-cm needle is used and is inserted 3-4 cm backwards slightly inwards and downwards During movement of the needle injection is continuous as a safeguard against injuring veins
- 2 Infero lateral from a wheal at the infero lateral margin of the orbit † Deposition of a little solution is also necessary in the superior rectus

If general anaesthesia is used bromethol will reduce the tension of the eyeball and so facilitate operation for squint Muscle relaxants are useful in these cases given in subhypnotic doses Otherwise barbiturates are the best premedication as morphine may produce vomiting after operation Heroin  $\frac{1}{4}$  gr as pre medication causes less vomiting than morphine

Macintosh R. R. and Ostler M *Local Anaesthetics Head and Neck* 1955 121 Edinburgh and London E & S Livingstone  
 † Atkinson W S *Anaesthesia in Ophthalmology* 1955 41 Springfield USA C C Thomas.



*Eye Surgery continued*

requires more nursing care and is slightly less safe than local analgesia. Ocular ischæmia is aided by a flawless anæsthetic technique: a tilted table and the instillation of adrenaline into the conjunctival sac. Intra ocular tension is reduced by the use of non depolarizing relaxants by lowering the blood pressure and by deep anæsthesia. A lowered intra ocular tension may be required for intra ocular lens extraction and for some other intra ocular operations: a normal tension for extra ocular lens extraction.

If local analgesia is to be used 4 per cent cocaine should be instilled into the conjunctival sac every two minutes on five occasions. This will give analgesia of the cornea and conjunctiva but not of the iris or ciliary body nor will it produce analgesia in a glaucomatous eye. It has the disadvantage that it produces dilatation of the pupil (bad in glaucoma) while it irritates and dries the corneal epithelium. Amethocaine 1 per cent when combined with adrenaline does not have these effects and can be used for tonometry. Holocaine butyn and piperocaine—all in 1 per cent solution—have also been used. Novutox can be used in the presence of sepsis, styes etc. for injection.

To block the iris and the globe of the eye making it insensitive and reducing its tension the ciliary ganglion and nerves must be blocked with 4 ml of 4 per cent procaine injected from a 3 in needle inserted from the middle of the upper lid to the roof of the orbit. For this retrobulbar injection a fine needle not longer than 1½ in should be used and should be inserted close to the bony margin of the orbit to prevent hæmatoma formation.

To immobilize the lids prevent screwing up the eyes and so raising intra ocular pressure twigs of the facial nerve are blocked in the parotid gland by injecting 3 c.c. of 1 per cent lignocaine on to the neck of the mandible just below the zygoma. If the eyeball is to be opened paralysis of the orbicularis oculi so produced is necessary to prevent squeezing of the globe.

Another method of making the eyelids insensitive and immobile so that a speculum can be introduced is to inject a little procaine from a wheal over the external palpebral ligament along the margin of each eyelid. From the same wheal the needle should be directed backwards towards the ear to block the fibres of the facial nerve supplying the orbicularis oculi.

Suxamethonium has been shown to cause contraction of the extra ocular muscles and increase in intra-ocular tension and it may well be that a non depolarizing relaxant is the one of choice in intra ocular operations.\*

**Local Analgesia for Surgery of Lacrimal Sac** †—Local analgesia reduces bleeding if adrenaline is added to any suitable analgesic solution e.g. 1 per cent procaine or 1 per cent xylocaine. The nerve supply of the lacrimal sac is from (1) The infratrochlear branch of the first division of the fifth nerve (2) The anterior

Dillon, J. B. Sabwala, P. Tylor, D. B. and Gubert, R. *Anæsthesiology* 1957 18 44

\* d 439  
† Philips, A. S. *Proc R Soc Med.*, 1951 44 169

Paravertebral block of T 10-L 2 is excellent for cases of strangulated hernia (see p 341) and regional block is also favoured in normal cases by some surgeons and anaesthetists (see p 357)

Post-operative chest complications frequently follow these operations especially in fit young men no matter what anaesthetic agent is used

They should consequently be advised to avoid smoking for the month preceding operation and be taught how to breathe deeply and how to cough effectively In addition they will require vigorous shake up treatment after operation

Operations for incisional hernia require deep relaxation

The chief danger in operation for strangulated hernia is from aspiration of stomach contents into the chest.

### **RADICAL MASTECTOMY**

An endotracheal tube minimizes oozing as also does a table tilt with head raised By the use of topical analgesia and short acting relaxants a smooth intubation free from coughing and straining should be carried out and this too will reduce the tendency to bleed Bromethol by reducing the blood pressure also reduces bleeding Only light general anaesthesia is required Gas and oxygen with minimal intermittent thiopentone and pethidine are a good combination with or without the addition of a little trilene Light cyclopropane tends to produce oozing

As shock may be produced an intravenous drip should often be put up at the commencement of the operation

### **GENERAL ANÆSTHESIA FOR AMBULANT PATIENTS**

This presents problems which may be most difficult to solve Factors to be considered are the need for rapid recovery and the lack of preparation of the patient e.g. his full stomach As in operations for major surgery anaesthesia should provide a quiet relaxed and unconscious patient The relatively unskilled anaesthetist may find cyclopropane and oxygen half of each useful given from a six litre bag followed if necessary by gas and 20 per cent oxygen Unconsciousness lasting from one and a half to two minutes can be produced in all patients by 10-15 breaths of the oxygen-cyclopropane mixture Vomiting is the disagreeable complication and is worse in children than in adults Nitrous oxide and oxygen alone or supplemented with trichlorethylene can be used Regular intake of alcohol is the great enemy of gas-oxygen anaesthesia Intravenous thiobarbiturates are useful Local analgesia is most rewarding in suitable cases

### **ACUTE INFECTIONS OF THE NECK**

These cases present difficulties because of possible —

1 Trismus

2 Voluntary use of accessory muscles of respiration to overcome respiratory obstruction associated with the lesion if this is present fatal hypoxia may follow loss of consciousness Thus tracheotomy under local analgesia may be necessary before the induction of general anaesthesia Blind nasal intubation in the conscious patient after spraying the nares and larynx with

**Eye Surgery—Retro ocular Block *continued***

Thiopentone is useful but may give rise to explosive coughing or sneezing during the operation. This is lessened but not abolished by the preliminary instillation of cocaine into the conjunctival sac. Thiopentone reduces the tension in the normal and glaucomatous eye. A silkworm suture through the tongue will give the anæsthetist considerable control over the airway while oxygen inhalations will add to the safety of the procedure. Some surgeons are using this technique for cataract extractions. But for delicate or prolonged operations an endotracheal tube should be passed after careful cocainization of the larynx and the injection if thought necessary of a muscle relaxant. Inhalation anaesthesia to Plane 2 can then be maintained quietly thus producing a still field with stationary globes and relaxed muscles. The thiopentone gas-oxygen trilene sequence is satisfactory.

For examination of the fundus in young children rectal thiopentone is excellent.

In eye surgery post operative vomiting must be reduced to a minimum.

Repeated small doses of dimethyl ether of *d* tubocurarine have been recommended\* for cataract surgery. The drug is given until ocular palsy shows itself. It causes general and local muscular relaxation allays apprehension and limits ocular movements preventing vitreous prolapse. Local but no general anaesthesia is used. Chlorpromazine, promethazine and pethidine—50 mg of each given intravenously will provide good sedation during cataract operations †.

During operations for squint in children the injection of 1 mg of *d* tubocurarine per stone helps the surgeon. Reflex cardiac arrhythmia has been reported during ocular muscle surgery especially involving the internal rectus. A full dose of atropine should be given before operation ‡.

**HERNIA**

The anaesthesia depends on the degree of relaxation demanded by the surgeon. Extradural block using 25–30 ml of 1.5 per cent lignocaine solution is suitable in fit subjects. If spinal analgesia is used block to T9 is necessary and is obtained by for example light nupercaine 10–12 ml 0.5 per cent nupercaine 1.6 ml procaine 150 mg. There may be discomfort when the sac is under tension this can be eased by the injection of a little procaine into the neck of the sac. Where much relaxation is not required light general anaesthesia is indicated and should the surgeon wish it a muscle relaxant can be injected together with such a combination as gas oxygen and thiopentone, cyclopropane, gas-oxygen and ether. Thiopentone, pethidine, gas and oxygen and flaxedil is also a very good combination.

Agarwal, L. P. *Br J Ophthalmol* 1953 7 558  
 † Ingram, H. V. *Br J Med* 1957 2, 222  
 ‡ Bosomworth, P. P., Zeigler, C. H. and Jacoby, J. *Anesthesiology* 1958 19 7 and Walton, F. A. *Canad Anaesth Soc J* 1957 4 414.

Paravertebral block of T 10-L 2 is excellent for cases of strangulated hernia (*see* p 341) and regional block is also favoured in normal cases by some surgeons and anaesthetists (*see* p 357)

Post-operative chest complications frequently follow these operations especially in fit young men no matter what anaesthetic agent is used

They should consequently be advised to avoid smoking for the month preceding operation and be taught how to breathe deeply and how to cough effectively In addition they will require vigorous shake up treatment after operation

Operations for incisional hernia require deep relaxation

The chief danger in operation for strangulated hernia is from aspiration of stomach contents into the chest.

### **RADICAL MASTECTOMY**

An endotracheal tube minimizes oozing as also does a table tilt with head raised By the use of topical analgesia and short acting relaxants a smooth intubation free from coughing and straining should be carried out and this too will reduce the tendency to bleed Bromethol by reducing the blood pressure also reduces bleeding Only light general anaesthesia is required Gas and oxygen with minimal intermittent thiopentone and pethidine are a good combination with or without the addition of a little trlene Light cyclopropane tends to produce oozing

As shock may be produced an intravenous drip should often be put up at the commencement of the operation

### **GENERAL ANÆSTHESIA FOR AMBULANT PATIENTS**

This presents problems which may be most difficult to solve Factors to be considered are the need for rapid recovery and the lack of preparation of the patient e.g. his full stomach As in operations for major surgery anaesthesia should provide a quiet relaxed and unconscious patient The relatively unskilled anaesthetist may find cyclopropane and oxygen half of each useful given from a six litre bag followed if necessary by gas and 20 per cent oxygen Unconsciousness lasting from one and a half to two minutes can be produced in all patients by 10-15 breaths of the oxygen-cyclopropane mixture Vomiting is the disagreeable complication and is worse in children than in adults Nitrous oxide and oxygen alone or supplemented with trichlorethylene can be used Regular intake of alcohol is the great enemy of gas-oxygen anaesthesia Intravenous thiobarbiturates are useful Local analgesia is most rewarding in suitable cases

### **ACUTE INFECTIONS OF THE NECK**

These cases present difficulties because of possible —

- 1 Trismus
- 2 Voluntary use of accessory muscles of respiration to overcome respiratory obstruction associated with the lesion if this is present fatal hypoxia may follow loss of consciousness Thus tracheotomy under local analgesia may be necessary before the induction of general anaesthesia Blind nasal intubation in the conscious patient after spraying the nares and larynx with

**Acute Infections of the Neck continued**

Local analgesic solution may be the method chosen by experienced workers in some cases. The inhalation of a mixture of helium 79 per cent and oxygen 21 per cent has a density of 330 as against 1000 for air. A volatile agent or cyclopropane can be added to this mixture and fairly readily inhaled with much less distress to the patient than he experiences with air.

Before inducing general anæsthesia in a patient with an acute infection of the neck who is hypoxic 100 per cent oxygen should be given for ten minutes followed by a smooth gas-oxygen-trilene induction. Early passage of a nasopharyngeal tube will remove any respiratory obstruction due to trismus or the presence of a bulky or œdematous tongue or pharynx. Blind nasal intubation helped by topical analgesia can then be carried out. A rather small tube e.g. size 4 or 5 is easier to insert than a larger one and is permissible for short operations. It is probable that thiopentone is not responsible for any excitation of carotid sinus reflexes in these cases. Thus in small doses for induction it is probably safe.

If the abscess is superficial it can be opened under refrigeration anæsthesia i.e. the application of ice to the part for 45-60 minutes.

**HÆMOPHILIA**

Plasma containing anti hæmophilia globulin—1 litre—should be infused and the operation done straight away.\*

**BURNS†**

These can often be cleaned and dressed under morphine given slowly by intravenous injection so that the minimal effective dosage can be ascertained.

Gordon has advocated the intravenous injection via a drip of 1 g of procaine dissolved in 500 c.c. of 5 per cent glucose saline.

After a time painless dressing of burns will usually be permitted. For dressing of burns gas and oxygen analgesia is useful.

**ANÆSTHESIA AND BLEEDING**

Some common causes of excessive bleeding—

- 1 Respiratory obstruction because it gives rise to (a) Hypoxia (b) Hypercapnia (c) Straining which raises the intrathoracic pressure during expiration and so increases venous pressure.
- 2 Coughing and bucking—for the same reasons.
- 3 Hypercapnia from hypoventilation excess of carbon dioxide in a defective closed circuit or added carbon dioxide.
- 4 Venous obstruction of the operative site e.g. in laminectomies bad posture e.g. faulty positioning of the head.

The following factors among others influence the degree of bleeding—

Leatherhead le R. A. L. *Anæsthesia* 1958 13 27 and K. kw. k. R. A. and W. H. P. *Lancet* 1957 1 647.  
 † See Rook, J. M. *Lancet* 1953 1 1214. Sh. n. on D. W. *Ibid* 1955 1 111. d. Middleton, H. G. and Wolfson, L. J. *Brit. med. Bull.* 1958 14 1.

- 1 The arterial blood pressure depending on the cardiac output and the peripheral resistance
- 2 Local venous pressure which may be influenced by gravity
- 3 Tone of capillaries which is related to the blood oxygen and carbon dioxide levels
- 4 Efficiency of the clotting mechanism

Bleeding and clotting time are slightly prolonged by nitrous oxide shortened by chloroform ether and cyclopropane Thiopentone has no effect.

Myocardium is slightly depressed by chloroform and ethyl chloride these drugs causing reduced bleeding and cardiac output Cardiac output increased by ether and cyclopropane which also produce vasodilatation and consequent increase in bleeding during light planes of anaesthesia As depth increases blood pressure falls and bleeding is reduced General anaesthesia increases the blood supply to the limbs at the same time that it decreases the supply to the splanchnic area The maximal effect is seen thirty minutes after induction\*

Closed or semi-closed anaesthetic circuits may by their resistance to breathing cause respiratory obstruction and increased bleeding A well-designed semi-closed circuit with a non re breathing valve is an excellent circuit for a patient who is breathing spontaneously with an adequate volume

Posture can influence bleeding while experimental work is being done on arteriotomy and subsequent retransfusion (Gardner W J *J Amer med Ass* 1946 **132** 572) and also on total spinal analgesia (Griffiths H W C and Gillies J *Anaesthesia* 1948 **3** 134) as means of minimizing bleeding Controlled arteriotomy was first employed in Britain by Bilsland in 1951 †

See also chapter on Hypotension in Surgery

### CONTROLLED HYPOTENSION BY ARTERIOTOMY

This technique is seldom used to day Blood withdrawn from the radial artery into bottles containing sodium citrate in amounts of 500 ml or less until the blood pressure is reduced to about 80 mm Hg and afterwards retransfused was first advocated by Gardner in 1946 The amount required to reach such a blood pressure varies between 300 and 3000 ml and it can be returned to the circulation when needed either before the end or at the end of the operation Intra arterial retransfusion by raising the coronary blood pressure and myocardial circulation soon raises the systemic blood pressure Haemorrhage probably liberates a hormone causing vasoconstriction

Mortimer‡ states that in cranial surgery controlled arteriotomy has four advantages (1) It makes bleeding more easily controllable because of hypotension and consequent vasoconstriction (2) It causes shrinkage of the cerebral cortex (3) It gives a relatively bloodless field in the vicinity of vascular tumours such as aneurysms of the circle of Willis (4) It reduces the need for blood donors See also Jack on I *Anaesthesia* 1954 **9** 13

Shackman R. and Graber I G *Br med J* 1952 **1** 14

† Bilsland, G W L *Anaesthesia* 1951 **6** 20

‡ Mortimer P L F *Ibid* 1951 **6** 128

**Acute Infections of the Neck** *continued*

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Lambert, R. A. L. *Anæsthesia* 1958 **13** 7 and Kwick R. A. and Wolf P. *Lancet* 1957 **1** 647.  
 † See Rook, J. M. *Lancet* 1953 **1** 1214. Shannon D. W. *Ibid.* 1955 **1** 111 and Middleton, H. G. and Wolfson, L. J. *Brit. med. Bull.* 1958 **14** 1 42.

post-operative state there may be diminished activity of the cough reflex which may predispose to atelectasis

In old age the most incurable of all diseases the emotions are often well under control so that the patient is easy to manage. Muscular relaxation is also often easy to produce. As metabolism is low sedatives should be ordered with care and atropine substituted for scopolamine as the latter may produce stimulation of the central nervous system with restlessness and delirium. It has been suggested that adverse cerebral effects of anaesthesia in old people are more common than is realized\*. Narcotics and sedatives should be used sparingly and should be limited to those such as paraldehyde which depress respiration least. Confusion caused by sedatives must not be treated by further sedation. Toothless gums and flabby cheeks may cause difficulty with the airway. Too much premedication may cause the patient to appear pale and collapsed. Hypoxia is not well tolerated.

Extra- and intradural analgesia is often very suitable for operations below the umbilicus. Blood pressure raising drugs should not be injected until it is known that the block is working well lest a blood pressure too high for the inelastic vascular system should be produced with a resulting vascular accident.

Thiopentone is well tolerated as a rule if dosage is kept down.

Early post-operation ambulation is usually desirable to prevent the development of phlebothrombosis while rapid recovery of the cough reflex is important in the prevention of post-operative atelectasis.

**Children**—In comparison with adults neonates have a larger proportion of dead space in their lungs. Their ribs are nearly horizontal in the position of deep inspiration while the diaphragm is pushed up by the large liver hence respiration is rapid diaphragmatic may easily become deficient. To compensate for this the infant's trachea is relatively wider in proportion to lung volume than is the adult's trachea so his shallower inspirations are facilitated. Deep inspiration is anatomically impossible in infants. The lungs are much less efficient ventilating organs than they eventually become with a respiratory surface per unit weight one third that of adults. To compensate for this the respiratory rate is increased in infants—up to about 40 per minute. Bucking does not occur in the first three months of life although the laryngeal reflexes are very active as the child is living on fluids. The blood volume of a newborn baby is about 500 ml and of a one year old double this. The normal blood pressure of an infant is about 80/60. Cyanosis in the newborn may reflect its high haemoglobin content. In infants the tongue is often pushed against the palate causing respiratory obstruction.

The child's larynx differs from the adult larynx in being † (x) Higher up the rima glottidis is opposite the third-fourth cervical interspace in the infant in adults one interspace lower

Beddo & P. D. *Lancet* 1955 2 259

† Eckenhoof J. E. *Anesthesiology* 1951 12 401



*Controlled Hypotension by Arteriotomy continued*

His technique is to induce anæsthesia and maintain it with cyclopropane. A brachial plexus block is performed to cause maximal dilatation of the radial vessels and to prevent their reflex spasm as part of a generalized compensatory vasoconstriction. A cannula is tied into the artery which is afterwards carefully sutured and through it blood is taken and given back as required.

On bleeding a time comes when vasopressor compensation reaches its limit and thereafter further bleeding causes a progressive hypotension.

\*     \*     \*

Choice of anæsthetic is always dependent on the skill and experience of the anæsthetist and on the preferences of the surgeon and the patient. Every form of anæsthesia has its disadvantages and complications and is responsible for a certain morbidity and mortality. To everything there is a season.

It is relatively easy to acquire techniques but judgement in their use is the reward of clinical experience carefully garnered (Walsh).

There is no general anæsthetic which can be called the best and an anæsthetist is likely to benefit his patient most when he uses the technique and drugs with which he is familiar. For the tyro there are few absolute contra-indications to ether which remains after 11 years the safest all purpose anæsthetic.

Skilfully managed any one of a number of agents and techniques can usually be applied the final solution often being a matter of individual preference.

## CHAPTER XXI

## CHOICE OF ANÆSTHETIC AGENTS AND METHODS AS INFLUENCED BY GENERAL CONDITION OF PATIENT

**Old Age.**—The following characteristics may occur: dehydration, tissue wasting, cardiac enlargement and perhaps dilatation, arteriosclerosis of renal, cerebral and cardiac vessels, stiffening of thoracic cage with ossification of cartilages, emphysema, narrowing of bronchioles and dilatation of alveoli, atrophy of brain and increase in cerebrospinal fluid volume with dilatation of cerebral ventricles, atrophy of mandibles and increased brittleness of bones, atrophy of tissue cells and proliferation of connective tissue, diminution of blood volume and hæmoglobin and decreased cardiac reserve with slowing of circulation time, decreased thoracic and increased abdominal respiration, decreased tidal volume and vital capacity with consequent tachypnoea. In the

useful for rapid induction and can if required follow nitrous oxide-oxygen. The open mask is usually better than the gas machine in infants.

Intubation is more frequently employed than it was before the use of relaxants made it relatively easy to perform. In very small babies however it may be safer to pass the tube while the child is still conscious and breathing so avoiding delay and difficulty in an apnoeic patient. A larger tube can be passed through the mouth than through the nose. Children up to one year seldom tolerate a tube larger than size 1. Children up to 5 years old will rarely take a tube larger than size 3.

In children an adult size reservoir bag is undesirable because in a large bag it is difficult to assess respiratory movements of low volume while the thick rubber may offer resistance to respiration. A latex bag of 500 ml capacity should be used.

The closed circuit should not be used unless specially designed apparatus is available to minimize dead space such as the absorber of Cope and that of Sandiford\*. The anaesthetist must remember the possibility of ether convulsions especially in hot weather or with pyrexial patients.

The tidal exchange in a newborn baby may be as little as 70 ml so even with a tiny mask dead space must be greatly increased unless oxygen is run under it to carry away excess carbon dioxide. Because of this small tidal exchange induction via an open mask may take a surprisingly long time. As the respiration rate is rapid the minute volume in children may be quite large so they require quite a large gas flow to prevent rebreathing in the ordinary semi-closed circuit.

In babies muscular relaxation requires neither deep anaesthesia nor relaxants. Protrusion of the intestine from the belly is due to diaphragmatic breathing and distension due to gas in the bowel not to the muscular tone of the abdominal wall. Deep anaesthesia should only be produced for short periods at critical stages of the operation and the child deliberately allowed to become light at other times.

A method of giving cyclopropane to infants by the semi closed circuit has been recently described†. After atropine pre medication anaesthesia is induced using a Boyle machine with Magill rebreathing attachment with the expiratory valve set at its slackest position. Oxygen 1.25 litres per minute (which remains constant throughout the anaesthesia) and nitrous oxide 3 litres per minute are given from a face mask and cyclopropane 750 ml per minute are added giving a cyclopropane percentage of 15. If deeper anaesthesia is required the nitrous oxide is reduced to 2 litres a minute and this increases the cyclopropane percentage to 20. The addition of a trace of ether vapour will increase relaxation.

Controlled respiration is often desirable as it relieves the respiratory muscles of work and prevents the gut from protruding. The rate

Sandiford H B C. *Anaesthesia* 1953 8 122

† France G G B & J *Anaesth* 1957 19 76

Choice of Anæsthetic in Children *continued*

(2) The epiglottis is relatively longer being V or Y shaped instead of flat as in the adult. It makes an angle of 45° with the anterior pharyngeal wall while in adults it lies closer to the base of the tongue. (3) The narrowest part of the larynx may be at the level of the cricoid cartilage which is not distensible as are the cords the narrowest part in adults. Thus an endotracheal tube may be squeezed through the glottis but be held up at the cricoid causing there trauma and œdema. If this occurs a smaller tube should be substituted and laryngeal trauma may be dangerous in infants and œdema may result either from clumsy intubation or from irritation due to too large a tube being left too long in the larynx. In children under 3 both main bronchi come off the trachea at an equal angle unlike the arrangement in adults\*.

## TIDAL VOLUME AND RESPIRATORY RATES IN CHILDREN†

Age	Total Volume	Rate
6 months	64 ml	55 per minute
12	79	45
18	131	33
2 years	143	30
3	156	31
4	180	30
5	192	28
6	199	27
7	204	30
8	228	25

*Premedication* after the age of two is important to avoid mental trauma. Rectal medication is resented by the young child so that pentobarbital by mouth  $\frac{1}{2}$  gr per st is a useful sedative. Otherwise bromethol 100 to 125 mg per kilo thiopentone 1 g per 50 lb or paraldehyde 60 min per st has a more certain effect. Morphine  $\frac{1}{8}$  gr with scopolamine  $\frac{1}{8}$  gr per st of body weight is a satisfactory premedication. An apprehensive child is likely to have persistent tachycardia throughout the operation especially if ether is given. Atropine and scopolamine must be given with great care to children with fever.

In infants a negative pressure is sometimes created in the stomach during inspiration and gas may be sucked in. To relieve this a catheter should be used as a stomach tube if the abdomen is distended or if breathing is laboured.

For induction a suitable method is to allow nitrous oxide to flow from a mask held 2 in above the patient's face. About 10 litres a minute are required for this. When unconsciousness supervenes the mask is applied to the face the gas flow reduced and oxygen added. Ethyl chloride and vinesthene are both

Bedford, P D *Lancet* 1955 2 259

† Hall, J E *P oc R Soc Med* 1955 49 761

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The tidal exchange in a newborn baby may be as little as 20 ml so even with a tiny mask dead space must be greatly increased unless oxygen is run under it to carry away excess carbon dioxide. Because of this small tidal exchange induction via an open mask may take a surprisingly long time. As the respiration rate is rapid the minute volume in children may be quite large so they require quite a large gas flow to prevent rebreathing in the ordinary semi-closed circuit.

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† France G. C. B. & J. *Anaesthesia* 1957, 19, 76.

Choice of Anæsthetic in Children *continued*

of ventilation should be rapid. The Ayre Y piece is very useful for this and is easier to manage than a closed circle with absorption of carbon dioxide. A B L B type bag open at both ends is fitted to the distal opening of a very short rebreathing tube while a vulcanite tap at the other end of the bag is so manipulated that intermittent pressure applied to the bag expels the amount of gas required to maintain the equilibrium of the system. The machine should deliver 3-4 litres/min of 50 per cent gas and oxygen with minimal ether if required. The pharynx should be carefully packed. Assisted breathing soon results in controlled apnoea. Deep anæsthesia must be avoided and occasional limb movements a sign of light anæsthesia can with advantage often be allowed.

For repair of hiatus hernia or tracheo-oesophageal fistula premedication should be atropine  $\frac{1}{2}$  g (0.3 mg). The upper air passages and oesophageal pouch should be aspirated before a tube is passed. N/5 saline is run into a suitable vein through a polythene catheter. Thiopentone 2.5 to 5 mg per lb is injected followed by either *d* tubocurarine 1 mg per 5 lb or suxamethonium 0.5 mg/lb. After insufflation with oxygen a number 00 Magill armoured tube is gently placed in the trachea. A fine catheter should be passed into the oesophagus via the nose to aid the surgeon. Maintenance should be by a mixture of equal parts of gas and oxygen and respiration should be controlled. A closed circuit e.g. Sandiford or Cope or a semi-closed circuit should be used. At the end of the operation neostigmine 0.03 mg/lb and atropine gr. 0.00 are given (unless suxamethonium has been used). Another method of anæsthesia is to insert a Magill armoured tube allowing ether-oxygen to flow down the smaller side tube while intermittent occlusion of the larger orifice results in assisted breathing. Some workers prefer to use oxygen and cyclopropane by the semi-open circuit. Intubation can be carried out in the conscious state. Small doses of relaxant may be desirable to facilitate controlled breathing.

Neonates should not receive too much fluid after operation and about 5 ml per hour is enough for the first two days later increasing\*.

**RELAXANTS IN CHILDREN**—Tubocurarine affects neonates like it affects myasthenic adults by increasing sensitivity and neostigmine is probably not a reliable antagonist†. In such patients it must be used with great care.

Suxamethonium is probably the preferred relaxant in neonates and they require at least twice the dose (dose for weight) to produce comparable results as in adults even though the pseudocholinesterase level is lower than in adults. A dose of 5 mg can be given and repeated many times if necessary. It sometimes causes bradycardia in infants.

*Doses for intubation* d tubocurarine 1 mg /5 lb (2-3 mg /stone)  
 Callamine 1 mg /lb Suxamethonium 0.5 mg /lb  
 (7 mg /stone) intravenously 1 mg /lb with hyaluronidase  
 intramuscularly 2 mg /lb without hyaluronidase intra-  
 muscularly

*Dose of neostigmine* 0.03 mg /lb (0.5 mg /stone) with atropine  
 at least  $\frac{1}{16}$  gr (0.3 mg) gr  $\frac{1}{8}$  (0.65 mg) if over 1 year

*Dosage of Intravenous Agents*—Thiopentone 2.5 to 5 mg /lb  
 (35-70 mg /stone) Pethidine 0.1 to 0.5 mg /lb

A relaxant can be very useful in a child with tachycardia tachypnoea and fever e.g. in a case of acute appendicitis or peritonitis which does not tolerate deep ether too well. In neonates a mixed or dual block is apt to develop after suxamethonium and can be antagonized by neostigmine. To reduce the total amount of drug intermittent injections are better than the continuous drip.

Hyperventilation may occur during anaesthesia in children and may be due to (1) Carbon dioxide excess (2) The stimulating effect of ether on the respiratory mucosa (3) Metabolic acidosis consequent on fever starvation etc. The first is relieved by cutting down the dead space and washing away excess carbon dioxide with oxygen. To avoid hydraemia infused fluid should not exceed 10 ml per lb in 24 hours.

Beware of loose teeth in children between 4 and 8 years of age.

For post-operative sedation papaveretum  $\frac{1}{8}$  g per stone of body weight is useful and efficient (i.e. 1 ml per stone of a mixture of papaveretum  $\frac{1}{4}$  g made up to 8 ml with saline). As a sedative and hypnotic for young babies chloral hydrate is beneficial the dosage being 2 gr for an infant 3 gr at 6 months old and up to 10 gr for older children. (See excellent article by G. Jackson Rees *Brit med Bull* 1958 14 1 38.)

**Pregnancy**—Anaesthetics are well tolerated but should if possible be avoided during the menstrual epochs in the early months lest anaesthesia gets the blame for possible abortion. Curare does not increase uterine tone in pregnancy nor do intradural and extradural blocks. Hypoxia and hypotension must be avoided.

**Asthma**—Anaesthetics well tolerated. Ether need not be avoided while small doses of bromethol are often well tolerated. Thiopentone not contra-indicated but must be used with care as both it and cyclopropane may cause bronchospasm and wheezing. Hexobarbitone is often preferable to thiopentone in asthmatic patients. In cases of severe bronchospasm the patient should receive bronchodilator drugs and sprays for several days before operation. The inhalation of a little ether vapour usually banishes the condition. Aminophylline 250-500 mg intravenously may also help.

**Anaemia**—Hypoxia must be avoided. Barbiturates reduce the number of circulating red cells by causing their absorption by the spleen. Cyclopropane is most satisfactory in these cases. If haemoglobin percentage is less than 50 a transfusion should be given. Extradural analgesia not suitable for patients with anaemia.

**Choice of Anæsthetic In Anæmia** *continued*

or who are likely to bleed during operation. Anæmia causes atony of muscles including myocardium. It also causes impairment of conduction of nerve impulses. In anæmia grave hypoxia may not be accompanied by cyanosis. There is an oxygen lack in the circulating blood—a reduction in oxygen content not in oxygen tension.

**Myasthenia Gravis** \*—This is a chronic disease of disputed aetiology characterized by exacerbations and remissions. It may occur at any period of life including childhood and old age and while severe cases are easily diagnosed mild ones can be overlooked and many cause anæsthetic difficulties. Some of the cranial nerves are usually involved and may give rise to ptosis, dysphagia and easy fatigue of the jaw muscles. Muscle weakness comes on after exercise and improves following rest. The myoneural block which is present may be non depolarizing in some end plates (and so reversible) but depolarizing in others. The myoneural junction may react to choline with the production of a non depolarizing block †. As a diagnostic test the intramuscular injection of neostigmine 1.5 mg with atropine 0.6 mg is useful and causes an increase in muscular strength in a few minutes. Edrophonium can also be used for this purpose. 2 mg are given intravenously and if improvement in muscle strength without fasciculation is not seen within one minute a second dose of 8 mg is given.

**PRE OPERATIVE PREPARATION**—The serum potassium should be estimated as hypokalaemia aggravates myasthenia. A chest radiograph is desirable. Respiratory infection should contraindicate all but the most urgent operations. Neostigmine should be given by mouth until the optimum dosage is reached. Sedative premedication should be minimal. Opiates and barbiturates should be avoided. Small doses of pethidine are suitable.

**ANÆSTHETIC MANAGEMENT**—The special concern is to ensure adequate respiration both during and after the operation while the special difficulties to be borne in mind are muscular weakness and bronchial secretion from neostigmine. Regional analgesia which does not depress respiration e.g. intra or extradural block to T10 may be suitable. Ether and thiopentone should be used most sparingly if at all but cyclopropane is a satisfactory agent. Relaxants are better avoided. The non depolarizers are contraindicated and it must be remembered that decamethonium may act as a non-depolarizer in this disease. Suxamethonium is the preferred relaxant but this too must be given as sparingly as possible. Some myasthenic patients are very resistant to suxamethonium and decamethonium. Endotracheal intubation may well be advisable both to ensure a perfect airway and to facilitate tracheobronchial aspiration. Neostigmine or edrophonium may be given intravenously with care either during or after operation if respiration is peripherally

depressed while aspiration of secretions from the upper respiratory tract must be performed with frequency, enthusiasm and efficiency. The possible need for artificial respiration perhaps from a machine and for tracheostomy must be provided for. In *carcinomatous neuropathy*, especially in carcinoma of the bronchus there may be a myasthenic response of the motor end plates and a consequent abnormal behaviour towards relaxants. Neuromuscular disorders e.g. weakness of limbs should be excluded in carcinoma patients before relaxants are given †

**Ankylosis of Jaw**—This may be part of a generalized arthritis and may render laryngoscopy difficult. It must be diagnosed before induction and sometimes blind intubation is of service.

**Euphroscoliosis**—These patients are likely to have reduced vital capacity so their tidal volume is limited and cannot increase e.g. in response to premedication which may slow the breathing rate. Underventilation in these patients is a danger even before anaesthesia is induced and may be present also after operation. They show respiratory handicap.

**Paraplegia, ‡**—Patients with transection of the cord at or above T 5 may get severe hypertension after certain visceral stimuli e.g. distension of the bladder or rectum. Hexamethonium may be required to relieve this.

**Dystrophia Myotonica §**—This rare disease is familial and its victims may show various signs of premature physical and mental senility including senile cataracts. It also may cause an abnormal response to thiopentone in that respiratory depression or even apnoea may outlast narcosis suggesting that the drug has a peripheral effect on abnormally sensitive muscles. This condition should be considered (as also should diabetes) in younger patients with cataract and if used at all thiopentone dosage should not exceed 100 mg. so avoiding prolonged respiratory depression.

**Liver Disease**—Chloroform and bromethol contra indicated. Thiopentone fairly well tolerated in small doses but as detoxication is largely in the liver large doses may easily cause prolonged narcosis. In addition large doses can actually cause additional damage to the liver cells. Excretion of pethidine delayed. Patients with a low value for pseudocholinesterase as is commonly seen in liver disease may require larger amounts of non depolarizing and smaller amounts of depolarizing drugs than normal patients. Liver function is often slightly depressed as a result of operation and anaesthesia. A progressive fall in the serum pseudocholinesterase which reaches its maximum about the fifth post operative day occurs and this suggests a mild dysfunction of the liver parenchyma.

Hiland, J. H. and Stephen, C. R. *Canad Anesth Soc J* 1958 5 323

† Croft P. B. *Br it med J* 1958 1 181

‡ Guttman L. and Whitteridge D. *Brain* 1947 70 36.

§ Bourke T. D. and Zuck, D. *Br J Anesth* 1957 29 135. Lodge A. B. *Br it med J* 1958 1 1043. Dundee J. W. *Curr Res Anest* 1952 31 257.



**Choice of Anæsthetic In Anæmia continued**

or who are likely to bleed during operation. Anæmia causes atony of muscles including myocardium. It also causes impairment of conduction of nerve impulses. In anæmia grave hypoxia may not be accompanied by cyanosis. There is an oxygen lack in the circulating blood—a reduction in oxygen content not in oxygen tension.

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heart disease than in normal patients a fact proved by the large number of successful operations performed actually for heart disease. Morphine and scopolamine have no serious effects on the heart. Two factors must be avoided in anesthetizing patients with heart disease—hypoxia and sudden hypotension. Adrenaline and similar sympatheticomimetic amines have a bad effect on the coronary circulation in diseases of these arteries: the larger branches are constricted, the smaller ones dilated. Hypoxia increases the coronary flow and hypercapnia has a similar though less intense effect.

Anesthetics may affect the heart

- 1 By interfering with the pacemaker and automatic conductive tissues
- 2 By their toxic effect on the myocardium
- 3 By depressing the peripheral circulation and reducing the venous return
- 4 By causing hypoxia which can result in arrhythmia and death from ventricular fibrillation in coronary disease, aortic valvular disease and in heart block

In addition post-operative chest infection, the intravenous infusion of too much fluid and thrombo-embolic complications may load the dice against the cardiac patient.

The cardiac patient should be made calm by his premedication so that nervous tachycardia which might cause heart failure is avoided. The orthopneic patient should be induced in his comfortable propped up position but can be made flat there after and pulmonary congestion and oedema is usually improved by good anaesthesia with its vasodilatation, muscular relaxation, decreased venous return and increased oxygenation.

- 1 **HYPERTENSION**—When regional analgesia is combined with adrenaline the latter must be used in minimal amounts. The diastolic blood pressure must be maintained. Induction should be by thiopentone with the possible addition of 5–10 mg of methylamphetamine to avoid cerebral and cardiac accidents in the stage of induction.
- 2 **HYPOTENSION**—Intra and extradural analgesia usually contra indicated if blood pressure is less than 100 mm Hg. Induced hypotension with the decreased coronary flow that it causes is rarely advisable in cardiac disease.
- 3 **DECOMPENSATION**—These cases are serious risks. The onset of acute pulmonary oedema is to be guarded against and if it shows itself properly treated e.g. a small dose of thiopentone with oxygen given under pressure. Spinal analgesia has also been used to treat the acute oedema. Intravenous fluid must not be allowed to overload the circulation. Intra and extradural analgesia with blood pressure supported with a continuous noradrenaline drip may suit some of these patients. Hypoxia must be definitely avoided. Thiopentone apart from small doses for induction must be used very carefully and combined with oxygen. Endotracheal intubation is often indicated. Cyclopropane is probably not contra indicated in these cases.

**Choice of Anæsthetic in Liver Disease** *continued*

Patients with liver disease including those with obstructive jaundice need several days of careful preparation before operation. Carbohydrates and protein should be given in large quantities. These patients should undergo a prothrombin time test to avoid overlooking a dangerous hypoprothrombinæmia. Prothrombin, a necessary link in the clotting mechanism, is synthesized by the liver under the influence of vitamin K, which is absorbed from the gut. As it is fat soluble, absence from the intestine of bile salts will give rise to poor absorption. Deficiency of the vitamin and so reduced prothrombin formation. If the prothrombin time is greater than 25 seconds or below 40 per cent of normal, the patient is a potential bleeder. Prothrombin deficiency is treated by the administration of synthetic vitamin K, either by mouth or by injection, a water soluble variety of the drug is used. Hypoprothrombinæmia due to vitamin K deficiency is the only type of this condition which responds to vitamin K therapy. If the state is due to decreased liver function or to liver disease, fresh blood transfusion is the correct treatment.

Severe prothrombin deficiency may exist in the absence of jaundice as synthesis of prothrombin by the liver depends on at least four factors: (1) Adequate amount of vitamin K in the intestine; (2) Presence of bile salts in intestine; (3) Normal intestinal absorption; (4) A healthy liver. Thus patients inadequately nourished, those with biliary fistula and those with pyloric obstruction may be potential bleeders.

Prothrombin concentration should be at least 75 per cent before operation is performed and some vitamin K must be continued post-operatively as long as prothrombin deficiency persists.

**LIVER FAILURE—**

- 1 There is delayed recovery from the anæsthetic, merging into semicomatose and death within forty-eight hours, preceded by hyperpyrexia. Post-mortem findings—necrosis of liver cells.
- 2 After four or five days, normal post-operative progress, patient becomes drowsy and comatose and dies, death being preceded by oliguria. Post-mortem—liver necrosis and kidney tubule necrosis.

Treatment is by intravenous amino acids and glucose.

**Kidney Disease—**Bromethol and thiopentone detoxicated more slowly in uræmia. Moderate doses of thiopentone may cause prolonged narcosis in such patients and those with a high blood urea. Gallamine excretion is delayed in patients with renal disease. Ether is a renal irritant.

**Cardiovascular Disease \*—**The risk of cardiac death during and immediately after operation is very little higher in patients with

1. Recent cardiac infarction
2. Angina pectoris. This has the following characteristics —
  - a. Its onset and cessation are related respectively to increased or decreased cardiac work.
  - b. Pain is usually retrosternal, not precordial.
  - c. Onset of a attack is sudden and duration short.
  - d. Nitroglycerine relieves pain quicker than cessation of activity would do.
 The pain of coronary occlusion is similar to the above but is more severe and long lasting, is not related to exercise nor relieved by nitrites.
3. Aortic stenosis
4. Syphilitic aortic reflux and aortitis with narrowing of the orifices of the coronary arteries.
5. Compensated heart block with Stokes Adams attacks
6. Large patent ductus arteriosus. The mechanism of possible collapse here is likely to be ventricular fibrillation. Hypoxia must be carefully avoided while pre-operative administration of 500-1000 mg. of procaine amide by mouth may be helpful. In case of emergency it can be given intravenously at a rate of 100 mg. per minute with 1000 mg. as the maximum dose. Hypotension may result.

Mitral stenosis with auricular fibrillation may lead to emboli formation after operation.

When digoxin is given to reduce the rate of the heart it acts by increasing the vagal tone, an effect abolished by atropine and ether. As atropine by paralysing the vagus increases the heart rate it may be undesirable in coronary disease.

The breath holding test of Sabrazès of Bordeaux (190 ) gives information as to the cardiorespiratory reserve. It is the time a patient can hold his breath after a normal expiration not preceded by a forced inspiration\*. A time of 20-25 secs. or longer is regarded as normal. If he gives up within 15 secs. he may present a serious risk. The breaking point is determined by the rising carbon dioxide tension of the blood.

Controlled hypotension when produced by ganglion block or high intra- or extradural analgesia is accompanied by vasodilatation so that the heart's work is reduced. When caused by arteriotomy (or hæmorrhage) there is vasoconstriction together with an increased load on the heart. As coronary disease cannot always be diagnosed controlled hypotension is potentially dangerous unless the operation cannot be done without it.

Thiopentone is a myocardial depressant and the handicapped heart muscle is less able to stand the insult than is a normal myocardium. In addition the drug causes peripheral vasodilatation while the tone of the heart may further be depressed by any associated hypoxia or hypercapnia resulting from the effects of thiopentone on the respiratory centre. Thus in heart disease the drug should be used most carefully and sparingly and should be given slowly in dilute solution.

Choice of Anæsthetic in Cardiovascular Disease *continued*

Pulmonary congestion predisposes to post-operative pulmonary morbidity. Before induction these patients are often benefited by an intravenous dose of digoxin (1 mg) or double that amount by mouth if they are not already taking it or one of its allies.

- 4 COMPENSATED HEART DISEASE—This is not a contra indication to intra and extradural analgesia below T 8-10
- 5 CONGENITAL HEART DISEASE—This must be regarded seriously. It may coexist with cleft palate etc
- 6 ACUTE RHEUMATISM—This should contra indicate all but emergency operations. Tonsils and adenoids should be removed during a period of quiescence
- 7 CARDIAC INFARCTION—A period of 2-3 months should if possible elapse between an attack and a surgical operation. In one series\* 18 per cent of patients died who were operated on during the acute stage
- 8 CORONARY DISEASE—Anoxia of the heart muscle must be avoided by maintaining an adequate diastolic blood pressure as coronary flow depends on the diastolic pressure. A turbulent second stage should be avoided. The mechanics of respiration must be safeguarded to ensure adequate filling of the right heart. The mortality rate of 517 patients with chronic coronary disease was 2.9 per cent compared with a rate of 2 per cent in 4154 patients with no clinical evidence of coronary disease.\*
- 9 AURICULAR FIBRILLATION—Should if possible be controlled before operation by digitalis quinidine etc. It is not a great hazard if the ventricular rate is well controlled. Frequent ectopic beats may require procaine amide.

In dealing with patients who have heart disease and who are about to have an operation and anæsthetic the following six questions must be answered† —

- 1 Will the operation and anæsthetic overtax the heart beyond its limits precipitating congestive failure?  
Does the heart require treatment before operation?
- 2 Is the cardiac prognosis so grave that the operation needs to be postponed or limited to a palliative proceeding?
- 3 Is the cardiac condition likely to lead to sudden death under anæsthesia?
- 4 What bearing does the state of the heart have on the choice of anæsthetic?
- 5 What if any cardiovascular complications are likely to follow operation or occur in the immediate post operative period?

Patients with organic heart disease who can carry on their daily jobs usually tolerate anæsthesia and operation well.

The following conditions should be detected before anæsthesia as they may lead to sudden death especially if hypoxia or sudden alteration in the blood pressure should occur —

Etsten, B. and Proger, S. *J. Amer. med. Ass.* 1955 159 845  
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Choice of Anæsthetic in Cardiovascular Disease *continued*

Gallamine may so increase the cardiac rate as to initiate decompensation so it must be used most carefully in heart conditions associated with tachycardia. A pulse rate above 130 per minute if prolonged may cause trouble.

Congestive heart failure occurring as a post operative complication may be difficult to diagnose. The appearance of triple rhythm or auricular fibrillation may give a clue while orthopnoea may precede signs of pulmonary congestion in early left heart failure. Increase in venous filling in the neck heralds right heart failure.

**Diabetes \***—Diabetes is a disease affecting the metabolism of fat and sugar and causes symptoms due to —†

- 1 A rising blood sugar—polyuria thirst pruritus etc
- 2 Ketosis i.e. acidosis causing hyperventilation loss of extra cellular sodium and water dehydration leading to circulatory collapse and coma
- 3 Peripheral vascular disease and neuritis. A severe diabetic is one who easily becomes acidotic if deprived of insulin usually the younger age groups—not the patient requiring large doses of insulin. During stress more insulin than normal may be needed.

When properly controlled the risk to diabetics is little more than to ordinary patients when they undergo surgical operations.

Regional analgesia gas and oxygen trlene cyclopropane thiopentone ether—are the choice of anæsthetics in approximately that order. Chloroform ether and bromethol must be avoided when possible as they cause a pronounced rise in blood sugar.

The diabetic may also have pulmonary tuberculosis.

The amount of glucose equal to the patient's carbohydrate requirement in twenty four hours is run in by intravenous drip and soluble insulin is given in divided doses four to six hourly depending on the amount of glycosuria or on blood sugar studies. Oral glucose is undesirable. It may not be absorbed and may cause vomiting. This scheme works well in major and emergency operations in diabetics of any severity and for minor surgery in bad diabetics or in cases difficult to control ‡.

*An alternative scheme of control*—In a diabetic who is not taking insulin but is cutting down carbohydrate an extra 50 g of glucose with 10 units of insulin should be taken each day for a few days preceding operation to re-stock the liver with glycogen. He can return to his normal habit a few days after operation.

Other patients should substitute for the meal before the operation 50 g of glucose with 25 units of insulin. If ether must be used an extra 10 units of insulin are needed in addition.

For emergency operations when the patient's diabetes is not controlled but when the ferric chloride test shows no diacetic

Foster P. A. and Francis, B. G. *Br J Anæsth.* 1955 27 291

† Cates, J. E. *Ibid* 1956 28 222

‡ King B. C. *Anæsthesia* 1957 12, 30.

acid (ketonuria) 50-100 g of glucose with 25 units of insulin should be given before operation but not by mouth for fear of regurgitation or vomiting with aspiration during anaesthesia. One pint of 5 per cent glucose contains approximately 25 g of glucose.

For emergency operations when ketonuria is present 100 g of glucose in 10 per cent solution with 100 units of insulin should be run into a vein until the urine becomes free from diacetic acid. Blood-sugar estimations are useful after operation but are not essential before emergency surgery.

There is a real danger of vomiting during induction in the diabetic with ketosis. Hexamethonium may enhance the action of insulin and cause severe hypoglycaemia. Diabetic patients sometimes develop a syndrome simulating an acute abdomen. The effects of soluble insulin last for four to six hours. Hyperglycaemic coma takes time to develop whereas hypoglycaemia can come on during an operation and may be characterized by sweating, pallor, tachycardia, dilated pupils, normal eyeball tension etc. It should be treated by intravenous glucose 25 per cent followed by 5 per cent solution and 25-50 units of insulin. It may be the cause of a delayed return of consciousness. Normal diet must be restored as soon as possible in the diabetic after operation.

#### **Pulmonary Disease —**

- 1 ACUTE —Inhalation anaesthesia is better avoided if it must be given cyclopropane or gas and oxygen are the most suitable while trilethylene is well tolerated. Penicillin or sulphonamides should be given to prevent post operative lung complications.
- 2 CHRONIC —Sputum should be coughed up before anaesthesia by postural drainage for two hours if necessary. Both cyclopropane and small intravenous doses of pethidine—a bronchial dilator—are fairly well tolerated. Thiopentone is not well borne if there is deficient aeration or if much sputum is present.

There are anaesthetists of skill and experience who do not regard acute and chronic lung disease as contra indications to the use of ether and when much bronchospasm is a feature ether maintenance following cyclopropane induction may go far to relieve the spasm and its resulting hypoxia.

*Chronic pulmonary emphysema* is a problem to anaesthetists and the following points merit attention: (1) Hypersensitivity of respiratory reflexes, bucking, bronchospasm etc. (2) Piston type respiration where diaphragm moves up and down like a piston in a cylinder while the thoracic cage fails to expand. This can seriously impede the abdominal surgeon in his work. (3) Dependence on chronic hypoxia for the respiratory drive via the aortic and carotid bodies. This is impaired if the patient breathes an oxygen rich atmosphere during or after anaesthesia. The pink colour due to this hyperoxaemia may mask hypoventilation and cause carbon dioxide narcosis. (4) Undue sensitivity to such respiratory depressants as opiates and barbiturates. (5) During controlled breathing there may be difficulty in inflation, reduced elastic recoil leading to inefficient

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There is a real danger of vomiting during induction in the diabetic with ketosis. Hexamethonium may enhance the action of insulin and cause severe hypoglycaemia. Diabetic patients sometimes develop a syndrome simulating an acute abdomen. The effects of soluble insulin last for four to six hours. Hyperglycaemic coma takes time to develop whereas hypoglycaemia can come on during an operation and may be characterized by sweating, pallor, tachycardia, dilated pupils, normal eyeball tension etc. It should be treated by intravenous glucose 25 per cent followed by 5 per cent solution and 25-50 units of insulin. It may be the cause of a delayed return of consciousness. Normal diet must be restored as soon as possible in the diabetic after operation.

#### **Pulmonary Disease —**

1 ACUTE — Inhalation anaesthesia is better avoided if it must be given cyclopropane or gas and oxygen are the most suitable while trilethylene is well tolerated. Penicillin or sulphonamides should be given to prevent post-operative lung complications.

2 CHRONIC — Sputum should be coughed up before anaesthesia by postural drainage for two hours if necessary. Both cyclopropane and small intravenous doses of pethidine—a bronchial dilator—are fairly well tolerated. Thiopentone is not well borne if there is deficient aeration or if much sputum is present.

There are anaesthetists of skill and experience who do not regard acute and chronic lung disease as contra indications to the use of ether and when much bronchospasm is a feature ether maintenance following cyclopropane induction may go far to relieve the spasm and its resulting hypoxia.

Chronic pulmonary emphysema is a problem to anaesthetists and the following points merit attention: (1) Hypersensitivity of respiratory reflexes, bucking, bronchospasm etc. (2) Piston type respiration where diaphragm moves up and down like a piston in a cylinder while the thoracic cage fails to expand. This can seriously impede the abdominal surgeon in his work. (3) Dependence on chronic hypoxia for the respiratory drive via the aortic and carotid bodies. This is impaired if the patient breathes an oxygen rich atmosphere during or after anaesthesia. The pink colour due to this hyperoxaemia may mask hypoventilation and cause carbon dioxide narcosis. (4) Undue sensitivity to such respiratory depressants as opiates and barbiturates. (5) During controlled breathing there may be difficulty in inflation, reduced elastic recoil leading to inefficient

*Choice of Anæsthetic in Cardiovascular Disease continued*

Gallamine may so increase the cardiac rate as to initiate de compensation so it must be used most carefully in heart conditions associated with tachycardia. A pulse rate above 130 per minute if prolonged may cause trouble.

*Congestive heart failure* occurring as a post-operative complication may be difficult to diagnose. The appearance of triple rhythm or auricular fibrillation may give a clue while orthopnoea may precede signs of pulmonary congestion in early left heart failure. Increase in venous filling in the neck heralds right heart failure.

**Diabetes** \*—Diabetes is a disease affecting the metabolism of fat and sugar and causes symptoms due to —†

- 1 A rising blood sugar—polyuria, thirst, pruritus, etc.
- 2 Ketosis, i.e. acidosis causing hyperventilation, loss of extra cellular sodium and water, dehydration leading to circulatory collapse and coma.
- 3 Peripheral vascular disease and neuritis. A severe diabetic is one who easily becomes acidotic if deprived of insulin, usually the younger age groups—not the patient requiring large doses of insulin. During stress more insulin than normal may be needed.

When properly controlled the risk to diabetics is little more than to ordinary patients when they undergo surgical operations.

Regional analgesia, gas and oxygen, trilethylene cyclopropane, thio pentone, ether—are the choice of anæsthetics in approximately that order. Chloroform, ether and bromethol must be avoided when possible as they cause a pronounced rise in blood sugar.

The diabetic may also have pulmonary tuberculosis.

The amount of glucose equal to the patient's carbohydrate requirement in twenty-four hours is run in by intravenous drip and soluble insulin is given in divided doses, four to six hourly depending on the amount of glycosuria or on blood sugar studies. Oral glucose is undesirable. It may not be absorbed and may cause vomiting. This scheme works well in major and emergency operations in diabetics of any severity and for minor surgery in bad diabetics or in cases difficult to control ‡.

*An alternative scheme of control*—In a diabetic who is not taking insulin but is cutting down carbohydrate, an extra 50 g of glucose with 10 units of insulin should be taken each day for a few days preceding operation to re-stock the liver with glycogen. He can return to his normal habit a few days after operation.

Other patients should substitute for the meal before the operation 50 g of glucose with 25 units of insulin. If ether must be used an extra 10 units of insulin are needed in addition.

For emergency operations when the patient's diabetes is not controlled but when the ferric chloride test shows no diabetic

\* Foster P. A. and F. acids, B. G. *Brit J Anaesth* 1955 27 292.

† Cates J. E. *Ibid* 1956 28 225.

‡ Lang B. C. *Anaesthesia* 1957 12 30.

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**Choice of Anaesthetic in Pulmonary Disease** *continued*

expiration and tendency to over inflation from trapping of air in lungs (6) Patient may be accustomed to a relatively high  $pCO_2$  (7) Tendency for neostigmine owing to its muscarinic effect to cause bronchospasm after operation

**PREOPERATIVE CARE**—This should include amelioration of bronchospasm and infection reduction in body weight and correction of anaemia

**REMEDIATION**—Promethazine and atropine are well tolerated. Opiates and barbiturates may cause undue respiratory depression. Pethidine in small doses is also valuable. Ephedrine and aminophyllin may be helpful

**ANÆSTHESIA**—Regional block e.g. extradural or brachial plexus when suitable. Good reasons can be advanced for preferring either spontaneous\* or controlled respiration †. If no relaxation is required spontaneous respiration with a smooth induction and perhaps a little ether or halothane avoiding too much oxygen so as to keep the respiratory drive active. When relaxation is required or when piston respiration makes life difficult controlled respiration may be necessary following the use of a relaxant. This may be followed by difficulty in restarting respiration. Hypoxæmia must be avoided and care taken with the  $pCO_2$ , hypercapnia may paralyse the respiratory centre while hypocapnia may fail to stimulate it if the tension is much less than that to which the patient has become accustomed. For bronchoscopy topical analgesia is often better than general anaesthesia.

Diffusion of nitrous oxide into gas filled body cavities e.g. pneumothorax may be quicker than diffusion of nitrogen outwards if nitrous oxide is inhaled. This increase in total gas pressure may be transmitted to the great veins causing cardiorespiratory difficulty ‡.

**Disease of the Nervous System**—Spinal analgesia and extradural block are usually inadvisable because future symptoms and signs may be blamed on these methods of analgesia. A history of frequent headaches may also make spinal analgesia undesirable.

**Alcoholism**—Alcohol addicts may require large doses of thiobarbiturates and volatile anaesthetics. Alcohol may well play a useful part in their premedication.

**Hypopituitarism**—Chronic hypopituitarism is most commonly due to ischaemic necrosis of the anterior lobe of the pituitary following hæmorrhage or shock in labour. It may be suspected if there is absence of a palpable thyroid, absence of pubic hair and genital hypoplasia. General anaesthesia is liable to precipitate coma in these patients. Cortisone may be helpful. Barbiturates and opiates too may have a very prolonged action §.

† J. J. L. *Brit. J. Anaesth.* 1956 33 165  
 ‡ Lunn J. *Brit. J. Anaesth.* 1958 30 114  
 § Hunt A. R. *F. & R. Soc. Med.* 1953 46 765  
 § Sherban H. L. *Br. J. Med.* 1953 2, 1022

## CHAPTER VIII

## SHOCK\*

What was formerly known as primary shock is now termed vaso-vagal collapse which is characterized by a slow pulse unlike the tachycardia associated with true (secondary) shock

The chief factor in shock is depletion of blood volume and replacement of that volume is the major therapeutic weapon. It has been defined by Wiggers as a syndrome resulting from depression of many functions especially reduction of effective circulatory volume and circulatory impairment progressing to circulatory failure. Irreversible shock has now come to mean shock which cannot be controlled by blood or similar fluid transfusion. Other factors may be the inability of the liver to inactivate the vasodepressor material circulating in the blood.

**Signs**—Pallor best seen in the face cold moist skin best seen in the extremities rapid weak pulse declining pulse pressure blood pressure drop is not an early sign collapse of superficial veins due to compensatory peripheral vasoconstriction and fall in venous pressure cyanosis of lips and nail beds thirst rapid shallow breathing vomiting restlessness diminished sensibility and subnormal temperature. The peripheral circulation time increases with fall in blood pressure whereas the increase in the pulmonary circulation time is relatively less.

**Causes**—

1. **HÆMORRHAGE**—A loss up to two pints is usually well compensated by splanchnic and cutaneous vasoconstriction. If bleeding continues true oligæmic shock is seen. Heart rate 100-120 which will probably be made faster by anaesthesia. In early stages hæmoglobin percentage is no index of blood lost. The decreased blood volume and the blood dilution cause a decreased venous pressure a decreased venous return and so a decreased cardiac output. Blood pressure is well maintained at first but falls later.

Should the patient with hæmorrhagic or wound shock not receive intravenous fluid he may

1. Become rehydrated from his own tissue fluids his cardiac output returning to normal but he remains anæmic. This is the hyperkinetic phase and in it rapid blood or fluid transfusion may overload the circulation and cause heart failure. A slow drip of packed red-cell suspension is required or—
2. He fails to rehydrate himself and passes into peripheral circulatory failure. A normal man has a blood volume of 5 litres.



**Choice of Anæsthetic in Pulmonary Disease** *continued*

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Johnson M. *Anæsthesia* 1956 11 165  
 † Gunn J F B & J. *Anæsthesia* 1958 30 14  
 Hunter A R. *Proc R Soc Med* 1955 48 765  
 § Sheehan H L. *Br J Med* 1955 2 1022

The lung manifestations are the most common

An early sign of fat embolism may be urinary incontinence. It has been said that collapse in the second hour after operation is likely to be due to shock, in the second day to fat embolism in the second week to pulmonary embolism.

**Air Embolism**—This may cause collapse or cardiac standstill. The patient should be turned on to his left side to relieve the air trap in the right ventricle. Cardiac massage may be required perhaps preceded by aspiration of air from the ventricles. The condition can often be diagnosed in its early stages by auscultation.

**Anæsthesia for the Patient in Shock.**—Operation should not be undertaken except in cases of grave emergency until the patient is resuscitated. Once this has occurred the longer operation is delayed the worse it is for the patient. The blood pressure should if possible be restored to at least 100 mm Hg and the clinical condition should show signs of improvement. A systolic blood pressure above 100 mm Hg indicates that blood volume is probably not less than 70 per cent of normal.

Shocked and recently resuscitated patients require much smaller amounts of anæsthetic agents than do normal patients and only light anæsthesia is usually necessary. Vomiting may occur at this light plane as shock is an important cause along with fear and anxiety of delayed gastric emptying.

Hypoxia must be avoided most carefully.

Premedication should be given intravenously just before induction of anæsthesia so that suitable dosage can be arrived at. On account of respiratory depression morphine may be contra-indicated.

Ether may cause theoretically increase in vasodilatation and capillary leakage. Actually light ether is a suitable anæsthetic. Cyclopropane is well tolerated. Should relaxation prove difficult a muscle relaxant can be satisfactorily combined with it.

Thiopentone is satisfactory if used in minimal doses but it must be remembered that in shock the distribution of thiopentone to non nervous tissues is interfered with so that removal of the drug from the blood stream has to be by the slow process of detoxication rather than by the more normal process of redistribution to fatty tissues. It should be combined with inhalation of either pure oxygen or mixtures of oxygen and nitrous oxide in equal proportion. A cuffed endotracheal tube is often desirable both to minimize the chances of underventilation and to guard against aspiration of gastric contents. Shock delays gastric emptying.

The blood pressure and pulse rate should be charted every five minutes. Some patients seem to benefit from an intravenous drip of noradrenaline (1:200,000) or the injection of 0.25–0.5 mg of ouabaine during operation (*see pp 279 and 473*).

**The Treatment of Shock before Operation**—Moderate warmth and rest. Elevate foot of bed. For restlessness and anxiety not due to pain give barbiturates e.g. pentobarbitone intravenously. Thirst may be more distressing than pain but is better relieved by

Causes of Shock *continued*

- 2 **TISSUE TRAUMA**—Circulating poisons the results of tissue autolysis or infection are absorbed into the circulation from damaged tissue. They injure the endothelial lining of the capillaries causing leakage of fluid into tissue spaces and producing a reduced blood volume. Sympathetic adrenal activity helps to compensate for this by causing vasoconstriction contraction of the spleen and cardiac stimulation. The peripheral circulation is reduced and extremities become cold and pale so that vital organs can be better supplied with blood. If condition is progressive venous return to heart and cardiac output are reduced and blood pressure falls. But this fall is not a sign of incipient shock rather it is a sign that the circulatory system is not able to cope with the emergency.

Owing to the poor circulation of the muscular and subcutaneous tissues absorption from them is delayed.

Renal failure may be due to the effect of these same toxins on the renal epithelium.

- 3 **NERVE TRAUMA**—Operative trauma may cause shock of sudden onset e.g. disarticulation of the hip joint rapid dilatation of a pregnant cervix uteri traction on the spermatic cord traction on the gall bladder or cardiac end of the stomach. Signs often disappear with the cessation of the stimulus. The anoci association theory of G. W. Crile is not now regarded as of major importance.

- 4 **BAD ANÆSTHETIC TECHNIQUE**—Overdose of anæsthetic or hypoxia can cause depression of the circulation.

- 5 **PROLONGED OPERATING TIME**

- 6 **ROUGH MOVEMENT OF THE PATIENT** while under anæsthesia.

Shock is also seen following prolonged or severe fluid loss (diarrhoea or vomiting) severe burns certain obstetrical manoeuvres coronary thrombosis perforation of certain viscera pleural puncture and introduction of certain foreign proteins into the tissues acute intestinal obstruction adrenal cortical insufficiency and certain toxins e.g. diphtheria.

The shock syndrome is not fully understood and views as to its exact nature and causation are fluid. Several of the above factors usually coexist to produce the syndrome clinically.

**FAT EMBOLISM**—This was first described in man by Zenker in 186 and may be wrongly diagnosed as shock. Due to escape of droplets of fat into the circulation and their deposition in the lungs brain or skin. Often associated with fractures of lower limb bones. Onset of symptoms may rapidly follow the injury or may be delayed for two or three days.

Pulmonary symptoms include dyspnoea pallor cyanosis pyrexia frothy sputum.

Cerebral symptoms may be restlessness leading to coma convulsions and paralysis. fat emboli may be seen in the retinal vessels with an ophthalmoscope.

Skin signs are likely to be a purpuric eruption with petechiae over the upper chest neck and conjunctivæ.

approached below the inguinal ligament where it lies medially to the artery. A sandbag should be under the infant's buttocks. In desperate cases the subclavian vein can be cannulated\* from a wheal raised just below the junction of the inner and middle thirds of the clavicle. A 3 in. needle attached to a syringe is inserted through the wheal backwards inwards and upwards hugging the under surface of the bone until the costoclavicular ligament is pierced with a dura like snap. A few millimetres beyond this point blood enters the syringe as the needle enters the vein. As hæmatoma formation leading to death has been known to follow the use of this method of intravenous medication it should be reserved for severe emergency when its use may be life saving.

To speed up the rate of flow often slowed down by the venous spasm associated with shock 10 ml of 1 per cent procaine solution injected into the tubing through an intradermal needle is useful. Nikethamide, 2 ml can also be used for this purpose. Raising the bottle well above the patient's limb may be helpful while air pressure can be raised in the bottle above the level of the fluid by means of a hand pump attached to the air vent tube. This last method must never be used if the level of fluid is below the top of the gauze filter for fear of air embolism.

A three way syringe can be used.

There is real danger in running blood too fast into patients with myocardial weakness (which may be due to anæmia the reason for the transfusion).

To make veins more prominent moist heat should be applied to whole limb for half an hour before venepuncture. Lundy suggests injecting saline with a small needle into a small vein applying a tourniquet so that larger veins become dilated with the saline. a large needle can then be inserted into this distended large vein.

For prolonged infusion a cannula should be tied into the saphenous vein at the ankle. This may predispose to phlebitis of the leg. Alternatively a polythene cannula can be inserted into the cephalic vein at the anterior axillary fold and passed up to a point just short of the angle in the vein where it passes through the clavipectoral fascia†. A similar tube can also be inserted into the superior vena cava from a small incision in the basilic vein 2 in. above and anterior to the medial epicondyl. Femoral vein transfusion may also be useful. Caval infusion‡ also has a place §.

To prevent thrombophlebitis from intravenous drips —

- 1 Use plastic rather than rubber tubing or silicone rubber or latex
- 2 Remove drip each eight hours

Aubalnac R. *Presse Méd* 1952 60 1456 Keeri Szanto M. and others *Canad Anas Soc J* 1957 4 55  
 † Jones P R. *P stigrad med J* 1957 33 446  
 ‡ Verel D. *Lancet* 1958, 1 716  
 § Chambers V W and Smith G. *Br J Surg* 1957 45 160.

### Treatment of Shock before Operation continued

mouth washes and intravenous fluid than by drinks if operation is pending. If necessary the stomach should be emptied by the passage of a large oesophageal tube.

Morphine should be injected intravenously and given only if the patient complains of pain. If given subcutaneously or intramuscularly absorption will be much delayed. This will render the drug useless as an anodyne and may result in sudden absorption of several doses on improvement of the peripheral circulation. Abdominal wounds are much more painful than thoracic. Only about one-quarter of seriously injured and shocked patients require morphine for pain relief and rarely should gr  $\frac{1}{4}$  be exceeded. It should not be given if there is severe brain injury or respiratory depression.

Transfusion of whole blood is of paramount importance especially if hæmorrhage has been marked. If the patient does not respond to intravenous blood transfusion an intra arterial transfusion should be set up as even 100-200 ml given by this route is likely to be helpful. A noradrenaline drip (2 ml of 1-1000 solution in 500 ml of saline) is a powerful vasoconstrictor which may raise the blood pressure even when whole blood fails. It should be given into a large vein directly or via an indwelling plastic tube pushed well up into a large vein to avoid possible tissue necrosis from intense vasoconstriction. If pale areas of skin develop injection into them of phentolamine 15 mg in 15 ml of saline with hyaluronidase may be helpful. Similarly injection into the skin of acetylcholine has been advised.

Oxygen inhalation not as useful as was once thought. Very useful in cases where oxygenation is deficient e.g. wounds of chest patients who are cyanosed. May be given if there is severe hæmorrhage tachycardia or respiratory depression.

If there is hæmoconcentration plasma or serum is indicated but two pints are usually sufficient except in cases of burn shock. Serum contains no fibrinogen and when reconstituted contains 14 per cent protein. Plasma contains 8-9 per cent of protein. These two are very useful in shock unassociated with much hæmorrhage e.g. after crush injury.

The amount of fluid given will depend on the patient's reaction to it. The aim is to restore the blood pressure to normal as quickly as possible. Each pint of blood should raise the systolic blood pressure 10-20 mm Hg and the hæmoglobin percentage 8 per cent.

**INTRAVENOUS INFUSION**—Suitable veins are the veins in the forearm or at the bend of the elbow. The saphenous vein anterior to the internal malleolus. Veins on the dorsum of the hand. Veins above the wrist. In infants the internal or external jugular or scalp veins can be used. For puncture of the internal jugular vein the child should be wrapped in a blanket enclosing the arms with its head overhanging the end of the table and rotated. A needle is inserted near the posterior border of the sternomastoid near its midpoint and advanced beneath the muscle perhaps as much as 1½-2 inches. The femoral vein is

Each blood transfusion carries a certain risk and among the possible hazards are the following —

1 *Hæmolytic reaction* —

*a Intravascular* Due to incompatible transfusion and the destruction of the donor's red cells by the action of specific antibodies in the recipient's circulation—chiefly anti A anti B and anti D of the Rhesus system. Usually ABO incompatibility causes a more violent reaction than one due to the Rhesus factor. General anaesthesia masks the effects. The blood pressure and pulse rate should be taken each five minutes for the first quarter hour with each new bottle of blood. If there is a fall of the blood pressure or a rise in pulse rate or cyanosis for which no other cause can be found the transfusion should be stopped.

*b Extravascular* Due to transfusion of lysed blood which is (i) Old (i.e. more than 3-4 weeks after collection) (ii) Heated above 38 C or (iii) Frozen (i.e. less than 4 C). The treatment of anuria due to incompatible transfusion demands the administration of 1000 ml of fluid daily through a stomach tube to which has been added an emulsion of arachis oil and glucose. The kidneys must be rested, alkalis and forced fluids are harmful. The idea is to prevent death from uræmia. When and if diuresis follows a careful watch must be kept on the electrolyte balance. If oligæmic shock develops compatible blood must be transfused and pressor drugs used. Many patients subjected to incompatible blood transfusion recover spontaneously if their kidneys have not been subjected to such insults as intravenous injections of citrate, lactate and sulphate of sodium.

2 *Use of infected blood*

3 *Circulatory overloading* —The rate of drip should be 0.5-1 ml per lb per hour. The signs are (a) Tightness in the chest (b) Cough (c) Dyspnoea (d) Cyanosis (e) Engorgement of neck veins (f) Tachycardia (g) Basal crepitations (h) Pulmonary oedema. Ventricular fibrillation has been reported after massive transfusion.\*

*Treatment* —Stop transfusion. Give digitalis and atropine. Prop patient up. Apply heat to dilate the skin vessels and enlarge the vascular bed.

4 *Air embolism* —The injection of 10 ml of air may prove fatal.

5 *Transmission of disease* —(a) Virus hepatitis (b) Malaria (c) Syphilis (d) Yaws (e) Relapsing fever (f) Kala azar.

6 *Febrile and allergic reactions* —Are said to occur in about 1-2 per cent of transfusions usually only mild urticaria resulting. Rarely asthma, angioneurotic oedema and laryngeal oedema are seen and must be treated by stopping the transfusion and giving adrenaline. The addition of

Treatment of Shock before Operation *continued*

- 3 Use a plastic cannula rather than a needle
- 4 Add heparin 1.5-2 units per ml of infused fluid This is insufficient to cause generalized bleeding

- 5 Add hydrocortisone 10 mg per litre to the infused fluid

**BLOOD TRANSFUSION**—First described in animals in 1666 by Richard Lower First successful man to man transfusion reported by J Blundell in 1829 Hustin of Belgium in 1914 demonstrated the usefulness of sodium citrate as an anti coagulant In 1900 Landsteiner first observed agglutination of human red cells by serum belonging to other individuals and described the ABO group The Rh system was discovered in 1939-40 (Landsteiner and Wiener) If the hæmoglobin percentage is less than 40 per cent a transfusion is necessary If a major operation is to be performed and the hæmoglobin is less than 70 per cent a blood transfusion is required A low value for hæmoglobin delays healing

Stored refrigerated blood (at a temperature of 4-6 C) is good for restoring hæmoglobin value and for raising blood volume up to about three weeks from the time of its collection but the clotting factor together with immune bodies deteriorate in stored blood Thus if the transfusion is planned in the treatment of sepsis hæmorrhagic disease or lack of clotting power blood less than twenty four hours old should be used

Rh negative blood should whenever possible be given to Rh negative patients who form about 15 per cent of the population in Britain It is specially desirable in —

- 1 Rh negative patients of either sex who have either had a previous transfusion or may require a subsequent one
- 2 Rh negative girls and women of child bearing age
- 3 Mothers of infants who have hæmolytic disease
- 4 Infants with hæmolytic disease

Packed red cell transfusion is carried out by siphoning off the supernatant fluid from two bottles of blood and adding the red cell deposits together It is useful in the treatment of anæmia before operation anæmia associated with myocardial disease and anæmia associated with nephritis By giving sedimented red cells three-quarters of the sodium content of whole blood is removed

Donor should be of the same ABO group as recipient while blood from an O donor should only be transfused into an O recipient in emergency Cross matching is also necessary except in emergency

**Direct Cross matching Test**—One drop of the recipient's serum is placed on a white tile and one drop of 5 per cent suspension of donor's blood in saline (2 or 3 drops of blood in 1 or 2 ml of normal saline) is added to it The two sera are mixed with a loop the tile tilted and left for 10 to 15 minutes and again agitated and examined with a hand lens for agglutination

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4 *Air embolism* —The injection of 10 ml of air may prove fatal.

5 *Transmission of disease* —(a) Virus hepatitis (b) Malaria (c) Syphilis (d) Yaws (e) Relapsing fever (f) Kala azar.

6 *Febile and allergic reactions* —Are said to occur in about 1–2 per cent of transfusions usually only mild urticaria resulting. Rarely asthma angioneurotic oedema and laryngeal oedema are seen and must be treated by stopping the transfusion and giving adrenaline. The addition of



Treatment of Shock before Operation *continued*

10 mg of chlorpheniramine (piriton) to each pint of blood reduces the incidence of reactions in allergic patients \*

- 7 *Thrombophlebitis*—This can be minimized by using plastic or silicone rubber tubing

Whole blood is citrated —

- a By adding to 420 ml of blood 100 ml of 3 per cent trisodium citrate in distilled water plus 20 ml of 15 per cent glucose in distilled water. Such blood must be used within fourteen days
- b By adding to 420 ml of blood 2 g of disodium citrate plus 3 g of glucose in 120 ml of distilled water. Such blood lasts twenty one days

*Citrate Intoxication*—The citrate in citrated blood is altered fairly rapidly into water and carbon dioxide and partly excreted as calcium citrate. Tetany can result from the removal of calcium ions in this way if a large amount of citrated blood (e.g. 4 litres in one hour) is transfused over a short period. A suitable ionizable calcium salt should be given after every 1500 ml of citrated blood given rapidly (e.g. 1 g of calcium gluconate). Cardiac depression may be caused by citrated blood due to (1) Citrate toxicity (2) Acidity of citrated blood. The pH is 7.1 when fresh (3) Removal of ionizable calcium by citrate (4) Hyperkalemia—in intra arterial transfusions (5) Paralysis of the cardiac vagus †. The oxygen released from blood stored in an acid citrate dextrose medium after transfusion may be decreased so the blood of an anæmic patient may for a few hours after transfusion be unable to release as much oxygen as it did before (see p 27). The potassium content of the plasma of stored blood is raised and should be remembered in cases where massive transfusions are given. Increased oozing may also occur.

**GLUCOSE** 50–100 ml of 50 per cent solution raises the blood pressure quickly and keeps it elevated for 20–30 minutes. Ordinarily 5 per cent glucose solution is used but if a combination of saline and glucose is to be employed the proportion should be  $\frac{1}{2}$  normal saline with 4.3 per cent glucose. This mixture is isotonic and will not irritate the veins. Glucose of 10 per cent or over may produce thrombosis. 5 per cent solution is isotonic and should be the routine solution for intravenous drips designed for fluid replacement and to keep open an intravenous route for medication.

**PLASMA** or serum is used in the liquid state or after adding distilled water to the dried powder. Plasma is prepared from citrated blood which does not clot and therefore contains fibrinogen. Serum is separated from blood which has been allowed to clot and so does not contain fibrinogen. Reconstituted plasma or serum should be used within three hours.

and not stored for future use. These substances are retained in the circulation, they do not leak through the capillary walls nor do they leave via the kidneys. Plasma is preferred to dextran as a blood volume restorer in conditions where a hypofibrinogenæmia is apt to develop e.g. in cases of accidental hæmorrhage, dead fetus in utero or hydatidiform mole. It may be necessary to use it in triple strength.

**POLYVINOYL BPC** (Plasmosan, Periston, Subtosan) — This is a 3.5 per cent solution of polyvinyl pyrrolidone in normal saline. It also contains ions of potassium, calcium and magnesium. It was suggested first by Hecht and Weese in Germany in 1943. It is a stable plasma substitute having a viscosity and colloid osmotic pressure similar to plasma. Is not antigenic and contains no protein. It is a substance foreign to the body. Does not interfere with blood clotting or antibacterial activity. Can be given through same apparatus as plasma but a saline wash through should be interposed between it and blood. Detectable in body up to two weeks after infusion although 75 per cent of it is excreted by the kidneys during the first ten days. It may interfere with interpretation of results of (1) Blood sugar, (2) Urinary albumin and (3) Erythrocyte sedimentation estimations. It does not interfere with blood grouping or cross matching. It is now seldom used in Britain.

**DEXTRAN** (Intradex, Dextraven, Macrodex, Expandex, Plavolex, Gentran) — A plasma substitute originally prepared in Sweden and suggested as a plasma expander in 1944 by Gronwall and Ingelman. It is a 6 per cent solution in normal saline of polydispersoid glucose polymer in which most of the molecules have been hydrolytically given a molecular weight similar to that of albumin. It is formed by bacterial action (*leuconostoc mesenteroides*) on sucrose. Its viscosity is between that of blood and plasma while its specific gravity is slightly greater than that of plasma. It contains antigens which occasionally give rise to skin sensitivity and other mild allergic lesions and can be ameliorated by the injection of antihistamine drugs. Such reactions do not detract from its usefulness. It is non-toxic, electrically neutral and chemically inert. Although it is eventually eliminated completely from the body it remains in the circulation in gradually decreasing amount up to a week and some of its larger molecules are stored for some time in the cells of the reticulo-endothelial system. Only 50 per cent can be recovered from the urine. The sulphuric ester of dextran has similar antithrombin activity to heparin which it structurally resembles. Ordinary dextran is not an anticoagulant. Has proved useful as a plasma substitute in cases of burns and surgical shock. It increases the venous return to the heart. During manufacture the size of its molecules must be made to approach that of plasma protein molecules. If too small dextran molecules pass through the glomerular filter, if too large kidney damage may occur. Some reactions occur in rather less than

Treatment of Shock before Operation *continued*

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Treatment of Shock before Operation *continued*

2 per cent of patients the chief being mild pyrexia. A few allergic phenomena are seen and very occasionally circulatory collapse, cyanosis and vomiting have been reported. Some samples may interfere with blood grouping and cross-matching results owing to rouleaux formation so specimens should be taken for this purpose before dextran is infused. If this is not done recipient cells must be washed before testing. It may interfere with plasma protein determinations. Blood sugar values not increased and diabetics tolerate it well. Can also be obtained in a salt free solution with 5 per cent dextrose and as a 10 per cent solution for use in cases of nephrotic and pregnancy oedema.

ACACIA 6 per cent (Bayliss 1917) in saline. PECTIN 5 per cent.

GELATIN 6 per cent have also been used.

Plasma substitutes cause no homologous serum jaundice (which occurs in 1.5 per cent of cases receiving small pool plasma). Up to 1 litre can be infused but larger amounts are not desirable. A pint of substitute to two pints of blood or plasma is a convenient and beneficial proportion. Their great advantage is their availability and stability in emergency especially in cases of (1) Acute hæmorrhage while waiting for blood, (2) Traumatic shock, (3) Burns, (4) Crush injuries, (5) Dehydration. They must be used carefully in cases of intestinal obstruction as they may be excreted into the bowel lumen when their osmotic properties are likely to withdraw fluid from the blood. They contain no nutriment are not oxygen carriers and are not buffering agents.

Whole blood is indicated —

- 1 In acute hæmolytic crises
- 2 In acute hæmorrhage
- 3 In subacute hæmorrhage which has led to secondary anaemia

Fresh blood is needed —

- 1 In prothrombin deficiency
- 2 In hæmophilia

Packed red cells are needed —

- 1 In anaemia of infection before operation
- 2 After acute hæmorrhage not treated by transfusion (the hyperkinetic phase of shock). Should be given within 24 hours of preparation to prevent infection.

Small pool plasma is indicated —

- 1 In acute hæmorrhage when blood is not available
- 2 In trauma and operation
- 3 In burns

Fresh plasma may be indicated in —

- 1 Prothrombin deficiency
- 2 Hæmophilia

Plasma expander may be indicated in —

- 1 Acute hæmorrhage when blood is not available
- 2 Trauma and operation
- 3 Burns

**THE RISKS OF OVER TRANSFUSION**—Harm is unlikely if the rate is slowed up when the blood pressure is back to normal or if this is not known to 100 mm Hg. The heart pumps out 5 l of blood each minute. Never more than 500 ml of fluid should be run in at speed under pressure.

In severe anaemia the pulse may be full and bounding, the pulse pressure high and the cardiac output increased in spite of a handicapped myocardium. In these cases large rapid transfusions may cause acute pulmonary oedema. Raising the head of the bed to encourage venous pooling in the legs together with intravenous digoxin 1 mg are useful should the heart begin to fail. One or two bottles should be given slowly or better still a transfusion of concentrated corpuscles should be given i.e. the corpuscles of two or more bottles are mixed after siphoning off part of the plasma.

Overload is dangerous too in patients with severe arteriosclerosis or coronary disease in whom a rise in pulse rate and fall in blood pressure during transfusion may signify developing heart failure rather than further haemorrhage.

Excessive amounts of plasma or serum dilute the blood and so produce anaemia.

Glucose and saline are not retained in the circulation so excessive amounts may cause acute pulmonary oedema owing to overfilling of vascular bed. For prolonged infusion in cases of dehydration forty drops a minute is a suitable rate giving one pint of fluid every four hours.

Thrombophlebitis following intravenous infusion is frequent if the drip remains up longer than eight hours. The nature of the rubber tubing used is an aetiological factor, silicone rubber and latex being less irritating than ordinary rubber.

**INTRAMEDULLARY INFUSION**—See Chapter VIII.

**HYDRODERMOCLYSIS**—This can be (1) Subpectoral (2) Into the outer side of the thigh (3) Into axilla (4) Into subscapular region (5) Into anterior abdominal wall especially in infants.

To prevent pain 1 g of procaine may be added to each litre of fluid.

Normal saline or 5 per cent glucose should be used.

**HYALURONIDASE**\*—This is a spreading factor which aids absorption of fluid injected into the subcutaneous and intra-muscular tissues. It is a mucolytic enzyme which hydrolyses hyaluronic acid, a viscous polysaccharide found in interstitial spaces which normally obstructs diffusion of invasive substances. It takes the line of least resistance and will not diffuse through an infected area because of the fibrin acting as a barrier. It is an enzyme first isolated by Mayer and Palmer in 1934 and described by Duran Reynals in 1929. It is a testicular extract prepared under the name of hyalase, rondase, wydase, diffusin, as a white powder, sterile and soluble in water. The contents of one ampoule 1 mg should be added to 500–1000 ml of fluid.

\* See article by W. Gausford and D. G. Evans *Lancet* 1949 2 505.

### Treatment of Shock before Operation *continued*

for hypodermoclysis the substance must be used as soon as possible after it is dissolved. It is non-toxic and has no side actions. An international standard of assay has now been defined by the W.H.O. at Geneva. It must not be contaminated with antiseptic which might inhibit its action.

**Uses**—(a) In paediatrics where veins are difficult to find. (b) In infiltration anaesthesia where it increases the area of effective analgesia. It does not increase the efficiency of nerve block and may make the infiltrated area painful in the hours following injection. No hyaluronic acid has been demonstrated in the extradural space and the enzyme is useless as an aid to extradural block. It is no substitute for precise anatomical knowledge but may be helpful in skin and subcutaneous analgesia, hernia block, splanchnic and pudendal block and in the reduction of fractures (e.g. Colles) under local analgesia. It has been reported to increase the toxic effects of local analgesia. (c) To maintain the fluid balance pre and post-operatively instead of intravenous infusions.

**PROCTOCLYSIS**—If a slow sustaining effect is required 5 per cent glucose in ordinary water is beneficial using a drip.

**Suprarenal cortical hormone** by reducing capillary permeability may do good in shock.

**PRESSOR DRUGS**—The intravenous injection—usually via a continuous drip—of sympathomimetic drugs has a part in the treatment of shock but conflicting opinions exist as to their proper place. Noradrenaline may redistribute available blood and may be useful in the periods when blood is not available. As the drug is a depressant of ganglionic transmission of vasomotor impulses the drip must be gradually withdrawn.

In spinal analgesia they are indicated as there is vasodilatation and absence of capillary leakage.

Excessive heat is contra-indicated in the treatment of shock.

**INTRA-ARTERIAL TRANSFUSION**—Intra-arterial transfusion was advocated in 1906 by Crile and Dolley and it was again advocated by Kemp in 1933. The left radial artery is cannulated 2 in. proximal to the styloid process under local analgesia or brachial plexus or stellate ganglion block to relieve spasm. Application of 1-40 papaverine solution also helps to release arterial spasm. The vessel is tied in continuity when the cannula is withdrawn. Apparatus may consist of two giving bottles connected in series with a Macintosh Pask drip chamber to prevent injection of air together with a bellows and manometer. In emergency a syringe and a 3-way tap may be used. **Indications** (1) When in severe shock the response to intravenous transfusion is poor. (2) During surgery when there is rapid and severe haemorrhage. The aorta may have to be used to receive blood. (3) When blood is in short supply.

The risk of gangrene is slight while it is usually no more difficult to cannulate an artery than a collapsed vein. use of

the high plasma potassium level in stored blood fresh blood should be used

**Use of Strophanthin G (Ouabaine)**—The injection intravenously of 0.5 mg. with or without replacement of blood volume improves the circulation in many patients and is useful if the response to fluid therapy is disappointing during before or after operation and as a preventative of circulatory depression during induction and maintenance of anaesthesia. The drug appears to do no harm acts quickly and for a short time and is rapidly excreted. It should not however be used in a patient who has received a digitalis glycoside within the past two weeks. Indications include† —

- 1 In shock as an aid to blood volume replacement  
To restore blood pressure when transfusion is undesirable e.g. in congestive failure
- 3 When there is peripheral vasoconstriction or tachycardia which might contra indicate pressor drugs
- 4 In sudden hypotension associated with induction of anaesthesia or movement of the patient
- 5 In congestive heart failure

#### **The Treatment of Shock during Operation —**

- 1 Lower head of table  
Lighten anaesthesia if necessary and see that oxygen is reaching the alveoli
- 3 Arrange for blood plasma or saline intravenous injection
- 4 Consider the use of 100 mg. of hydrocortisone and/or a nor adrenaline drip

### **SALT AND WATER BALANCE**

- 1 **Fluid Requirements**—Water balance is the ratio between the water taken in by all routes over the water leaving the body by all routes. Loss of fluid may occur independently of but more usually together with loss of electrolytes chiefly sodium chloride. The fluid balance is worked out as follows† —

<i>Intake</i>		<i>Output</i>	
As food	1100 ml	As urine	1500 ml
As drink	1500 ml	As faeces	100 ml
		Vapour from lungs	400 ml
		Vapour from skin	600 ml
Total 2600 ml		Total 2600 ml	

The healthy kidney can concentrate the day's waste products (average 35 g.) into 500 ml. of urine the specific gravity of which may be as high as 1032. Twice or thrice this volume of urine may have to be secreted to remove the waste solids if the kidneys are handicapped.

See articles by Horton J. A. G. and others *Brit med J* 1953 2 1249 Brown A. S. *Lancet* 1953 2 745

† Horton J. A. G. and Armstrong Davison M. H. *Br J Anaesth* 1955 27 139

‡ S. article by H. F. B. ewer (1949) "Blood Transfusion and Fluid Replacement Therapy *Modern Practice of Anaesthesia* (ed. Frank Evans) London Butterworth.



### Treatment of Shock before Operation *continued*

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The risk of gangrene is slight while it is usually no more difficult to cannulate an artery than a collapsed vein. Because of

addition the urinary chlorides are normal (3-5 g per litre)  
salt depletion is not serious

- c **TRANSFUSION**—Abnormal loss of fluid should be replaced by an equal volume of fluid to that lost in addition to the normal fluid requirements of the body. In exceptional cases as much as eight pints a day may be necessary e.g. ileus persistent diarrhoea

- i **ISOTHYLACTIC**—Tap water one and a half pints can usefully be given per rectum while patient is on the table as this volume of fluid is often lost during a major operation. It cannot cause overloading with either salt or water and is specially useful in children (in proportionately smaller amounts)

- ii **CURATIVE**—Water by mouth rectum vein or hypodermoclysis. Glucose 5 per cent in distilled water is an isotonic fluid useful for this purpose providing in addition 200 calories per litre. As salt loss frequently accompanies fluid loss one fifth normal saline with 4.3 per cent glucose can often be substituted. Normal saline containing 9 g of salt per litre should not be used in the absence of abnormal salt loss so that the kidneys are not presented with excess of salt to excrete renal impairment may result in oedema from salt retention should this occur

The average requirements after operation are as follows

First day  $3\frac{1}{2}$  l second and third days 3 l—by any available route. Too much fluid is harmful as pulmonary oedema and cardiac embarrassment may result

- 3 **Salt Requirements**—Basic need of body for salt is 1-2 g per day but as much as 15 g may be taken with food the excess being excreted in the urine (normal 5 g per litre). In cases of salt lack as soon as the plasma salt level falls below normal (560-630 mg per 100 ml) the kidneys cease to excrete salt

#### 4 **Salt Depletion.**—

- a **CAUSES**—Usually occurs when salt is lost but fluid intake is normal as in vomiting diarrhoea intestinal fistulae or drainage intestinal obstruction etc

- b **SYMPTOMS**—Lassitude apathy weakness anorexia vomiting peripheral circulatory failure. Urine normal in amount

#### c **RESULTS**—

- i A loss of total osmotic pressure of extracellular fluid leading to excretion of water by the kidney and thus to decreased extracellular fluid volume with secondary or extracellular dehydration. Later follows a fall in plasma chlorides with a low value for urinary chlorides as shown by Fantus's test. There may be a rise in blood urea

- ii There is a disturbance of acid base balance if the loss of chloride and sodium ions is disproportionate. With continued loss of gastric juice there is hypochloremia with the production of alkalosis which may be accompanied by a rise in the plasma bicarbonate level as the freed sodium combines with carbonic acid. On the other hand with continued loss of fluid from the

### Salt and Water Balance—Fluid Requirements *continued*

If water depletion is suspected a fluid balance chart should be kept. The following three types of water and salt depletion are described:

- 1 No liquid intake e.g. oesophageal obstruction treatment is plenty of water only a little salt
- 2 No intake plus loss of secretion e.g. pyloric obstruction treatment is plenty of water plenty of salt
- 3 Normal intake with loss of secretion e.g. diarrhoea treatment is little water plenty of salt

**2 Fluid Loss**—When the patient appears clinically to be dehydrated about 6 per cent of his body weight in water has already been lost and this amount must be replaced to restore hydration.

#### a CAUSES—

- 1 Inability to ingest adequate fluid after operation and anaesthesia conia severe prostrating illness dysphagia
- ii Loss of fluid from alimentary canal Seven and a half to ten litres of fluid are daily excreted into the gut and reabsorbed into its more distal segments. This gastro-intestinal fluid circulation is two or three times the volume of the average amount of fluid taken by mouth in twenty-four hours. It is thus seen that vomiting suction drainage diarrhoea intestinal fistulae etc. may have a powerful effect on the fluid economy.

Disturbances in this circulation have been given by Nadler as follows—

- 1 Removal of fluid from proximal gut e.g. vomiting gastric and duodenal fistulae high intestinal obstruction intestinal suction drainage
- 2 Failure of secretions to reach absorptive area as in ileus acute dilatation of stomach intestinal obstruction
- 3 Escape of secretions and wastage e.g. biliary drainage pancreatic and intestinal fistulae
- 4 Increased peristalsis leaving insufficient time for reabsorption as in diarrhoea

These fluids are isotonic with serum so salt is lost too.

- iii Excessive sweating which may cause great fluid loss. Sweat contains 0.2 per cent sodium chloride so loss of one litre of it produces a loss of one litre of water the excess salt (0.89 per cent minus 0.2 per cent) remaining in the body until it is excreted by the kidney because there is not sufficient water to support it as normal saline in the body. The salt is nevertheless excreted so the one litre of sweat results in loss of one litre of normal saline. Treatment is normal saline.

#### b DIAGNOSIS—On clinical and laboratory evidence

**CLINICAL**—Thirst dryness of mouth because of scantiness of saliva and oliguria. Thirst is more characteristic of fluid than of salt depletion.

**LABORATORY**—Raised blood urea because urinary volume is not sufficient adequately to carry away the non-protein nitrogen. High specific gravity of urine. If one pint of urine is passed each eight hours and is of low specific gravity all is probably well as far as fluid balance is concerned. If in

addition the urinary chlorides are normal (3-5 g per litre)  
salt depletion is not serious

- c **TREATMENT**—Abnormal loss of fluid should be replaced by an equal volume of fluid to that lost in addition to the normal fluid requirements of the body. In exceptional cases as much as eight pints a day may be necessary e.g. ileus persistent diarrhoea.

- i **PROPHYLACTIC**—Tap water one and a half pints can usefully be given per rectum while patient is on the table as this volume of fluid is often lost during a major operation it cannot cause overloading with either salt or water and is specially useful in children (in proportionately smaller amounts)
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- c **RESULTS**—
- i A loss of total osmotic pressure of extracellular fluid leading to excretion of water by the kidney and thus to decreased extracellular fluid volume with secondary or extracellular dehydration. Later follows a fall in plasma chlorides with a low value for urinary chlorides as shown by Fantus's test. There may be a rise in blood urea.
- ii There is a disturbance of acid base balance if the loss of chloride and sodium ions is disproportionate. With continued loss of gastric juice there is hypochloræmia with the production of alkalosis which may be accompanied by a rise in the plasma bicarbonate level as the freed sodium combines with carbonic acid. On the other hand with continued loss of fluid from the

Salt and Water Balance—Salt Depletion *continued*

gut distal to the pylorus there is a greater loss of sodium ions with resulting acidosis and decrease of the plasma bicarbonate. The alkali reserve normally 55–75 ml of carbon dioxide per 100 ml of plasma may be over 100 ml or less than 20 ml after vomiting or diarrhoea respectively.

- d **DIAGNOSIS**—On the clinical signs and symptoms. On the urinary chloride estimation or test of Fantus.

**FANTUS'S TEST**—Ten drops of urine are accurately measured into a test tube with a pipette. After washing out the pipette one drop of 20 per cent potassium chromate solution is added and the pipette rinsed again. Next 2.9 per cent solution of silver nitrate solution is added drop by drop the test tube being shaken after each drop. The end point is a sharp colour change from yellow to brown and the number of drops needed to produce it gives the concentration of sodium chloride in the urine expressed as grammes per litre. 5 drops i.e. 5 g per litre is normal.

If the end point change occurs with the first drop chloride is absent. If the urinary specific gravity is 1.020 anything less than 3 g per litre suggests salt depletion. If it is 5 g per litre and the patient is not suffering from Addison's disease and is not receiving intravenous saline he is probably not short of salt.

This test assumes the presence of relatively normal kidneys. If this is not so plasma chloride estimations are required. Its accuracy and usefulness have recently been called in question.

The combination of a high urinary chloride with a low plasma chloride should suggest the possibility of potassium deficiency which can be corrected by giving Darrow's solution intravenously or potassium chloride by mouth. The body's potassium needs are 2 to 4 g per day. If supplemented intravenously electrocardiographic control is desirable.

- e **TREATMENT**—Normal saline by any suitable route. When the symptoms are relieved and the urinary chlorides return to normal one fifth normal saline with 4.3 per cent glucose can be substituted. Coller, Bartlett and Maddock advise the administration of 3.2 g of salt per stone of body weight for every 100 mg that the plasma-chloride level needs to be raised to normal (560 mg per cent). Normal saline is suitable for both types of acid base disequilibrium.

The gastric solution and the intestinal solution of Cooke and Crowley\* are simple solutions designed to replace fluid loss. If this takes place above the pylorus from vomiting or gastric suction an acid medium with chloride ions in excess of basic ions is lost from the body. If it takes place below the pylorus the loss of bile and pancreatic juice by fistula or drainage will remove an alkaline fluid with base in excess of chloride.

The gastric solution contains 17 mEq per litre of potassium  
63 mEq per litre of sodium  
130 mEq per litre of chlorides (the  
excess 70 mEq of chlorides  
being neutralized by ammonium)

The intestinal solution contains 12 mEq per litre of potassium  
138 mEq per litre of sodium  
100 mEq per litre of chlorides—  
excess 50 mEq of base  
neutralized with lactate

These solutions are isotonic and can be given intravenously or hypodermically and if the appropriate solution is given in volume equivalent to the fluid lost there should be no great change in the electrolyte composition of the body

It is wise before undertaking fluid therapy to assess thoroughly the following three points —

- 1 Is fluid really necessary?
- 2 What fluid should be given?
- 3 What quantity of the correct fluid should be given?

(See also excellent paper by L. Le Quesne in *The Management of Abdominal Operations* (ed Rodney Mangot) 2nd ed 1957 London H. K. Lewis)

## CHAPTER XXIII

### ANÆSTHESIA IN THORACIC SURGERY

**Development**—In trying to avoid atelectasis Sauerbruch did his early thoracotomies in an airtight chamber with atmospheric pressure reduced by 7 mm Hg while the patient's head and the anaesthetist were outside in atmospheric air (negative pressure breathing). He later advocated positive pressure breathing. Lisberg obtained the same effect in 1910 by insufflating anaesthetic gases into the trachea at a positive pressure of 20 mm Hg. The first use of a bronchus blocker was by Archibald at the suggestion of Harold R. Griffith in 1935. Crafoord of Stockholm reported his method of artificial respiration by means of Frenckner's mechanical spiro pulsator in 1938 and this was developed and simplified by Nosworthy (1941) who advocated controlled breathing by intermittent pressure on the reservoir bag of a closed circuit using cyclopropane. This technique had previously been introduced by Guedel and Treweek in 1934 using ether. Cyclopropane had a great popularity during the decade following 1935 but is now on the decline in chest surgery because of its effect on the automatic conducting tissue of the heart and because of the increasing use of diathermy with the risk of explosion. The use of muscle relaxants has made it relatively easy to control respiration.

Salt and Water Balance—Salt Depletion *continued*

gut distal to the pylorus there is a greater loss of sodium ions with resulting acidosis and decrease of the plasma bicarbonate. The alkali reserve normally 55–75 ml of carbon dioxide per 100 ml of plasma, may be over 100 ml or less than 20 ml after vomiting or diarrhoea respectively.

- d **DIAGNOSIS**—On the clinical signs and symptoms. On the urinary chloride estimation or test of Fantus.

**FANTUS'S TEST**—Ten drops of urine are accurately measured into a test tube with a pipette. After washing out the pipette one drop of 20 per cent potassium chromate solution is added and the pipette rinsed again. Next 2.9 per cent solution of silver nitrate solution is added drop by drop the test tube being shaken after each drop. The end point is a sharp colour change from yellow to brown and the number of drops needed to produce it gives the concentration of sodium chloride in the urine expressed as grammes per litre. 5 drops i.e. 5 g per litre is normal.

If the end point change occurs with the first drop chloride is absent. If the urinary specific gravity is 1020 anything less than 3 g per litre suggests salt depletion. If it is 5 g per litre and the patient is not suffering from Addison's disease and is not receiving intravenous saline he is probably not short of salt.

This test assumes the presence of relatively normal kidneys. If this is not so plasma chloride estimations are required. Its accuracy and usefulness have recently been called in question.

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Premedication may consist of atropine in large doses and neostigmine—1.5 to 2.5 mg—and cyclopropane or thiopentone in small amounts. Gas oxygen and pethidine are suitable anaesthetics but an endotracheal tube although disliked by Keynes as it may cause tracheitis makes the anaesthetic safer and easier. Anti-depolarizing relaxants are contra-indicated as they prolong muscular weakness by interfering with the action of acetylcholine at the end plate. Depolarizing relaxants may act like anti-depolarizing relaxants in this disease and may be reversible by anticholinesterases. They may too prolong muscular weakness but small doses of decamethonium or suxamethonium used for intubation or to obtain control of respiration are unlikely to cause prolonged respiratory depression. Some patients have an abnormal resistance to their action. Respiratory depressants should be avoided. Neostigmine and atropine given after operation can be repeated in two hours if the respirations are shallow.

Special post-operative risks include —

- 1 Atelectasis—owing to interference with coughing because of muscular weakness and interference with the physics of the thorax.
- 2 Excessive mucus secretion due to neostigmine or pyridostigmine. This is controllable by atropine.
- 3 Pneumothorax due to surgical accidents. If respiration continues inadequate the patient should be treated like a case of bulbo-spinal poliomyelitis by tracheostomy, a cuffed tube and intermittent positive pressure respiration.\*

**CRUSH INJURY OF THE CHEST**—This may cause painful breathing or subsequent hypoxia and cardiorespiratory embarrassment. Injury (and operation) to the chest wall is likely to interfere with the patient's ability to rid the tracheobronchial tree of secretions. Moisture accumulates in areas of lung underlying the traumatized area.

To help the patient to breathe and cough intravenous procaine—500 ml of 0.2 per cent solution—is useful†. It will often prevent the development of traumatic wet lung. Adequate strapping and intercostal block may also be helpful. A patent airway must be maintained using suction, endotracheal intubation or tracheotomy. The control of paradoxical movements of the chest wall may be managed by stabilizing a portion of the thoracic cage by skeletal traction or by intermittent positive pressure respiration through a tracheostomy using a muscle relaxant‡. The cases can also be managed by intermittent positive pressure respiration after a tracheostomy has been performed (to aid suction and to reduce dead space) apnoea being induced by hyperventilation§.

Ch'ng J. and others *Canad. Anaest. Soc. J.* 1957 4 13.

† Rook J. R. *Anaesthesia* 1951 6 221.

‡ Bernatz P. E. and others *Proc. May Clin.* 1953 28 193.

§ Avery A. A. *J. thorac. Surg.* 1956 32 291.

**A Operations not requiring Pneumothorax.—**

**THORACOPLASTY**—Good results follow regional or general anæsthesia or a combination of both if the methods are applied with skill. Regional analgesia (*see p 340*) with or without thiopentone minimal chloroform gas oxygen trilene cyclopropane or halothane extradural block (*see Chapter XVII*) thiopentone and gas and oxygen.

The advantages of local analgesia over general anæsthesia in thoracoplasty are said to be (1) Reduction of hæmorrhage (2) Reduction of movement of lungs and intrathoracic structures (3) Reduced risk of spread of disease (4) Better elimination of secretions as cough reflex is not abolished (5) Quicker convalescence because patient is less upset by drugs used and needs less nursing care (6) Abolition of explosion risk. (H Bruce Wilson)

Good results follow the use of thiopentone gas and oxygen and a muscle relaxant such as gal amine to control such reflexes as phonation coughing laryngeal spasm and apnoea during apicolysis which might result in tearing of a thin pleura. In cases with sputum an orotracheal tube should be used and the head kept well down. Assisted respiration may be required after the apical parietal pleura has been freed. To reduce the amount of anæsthetic needed the line of incision and the intercostal nerves may be infiltrated with lignocaine 0.5 per cent solution or amethocaine solution (1-2000 to 1-4000). The risk of producing tuberculous laryngitis by intubation in patients suffering from tuberculosis is probably not great.

Although the pleura is not opened in thoracoplasty paradoxical breathing can occur as the support of the thoracic cage is lost. The same holds for thymectomy when the sternum is split. Adequate support of the chest wall by strapping is necessary after operation.

**PERICARDIECTOMY AND CARDIOLYSIS**—Frequently done under cyclopropane anæsthesia. Pericardiectomy often involves opening of the pleural cavity. Thiopentone should be used with great caution if at all in patients suffering from constrictive pericarditis.

**THYMECTOMY**—Pioneers in the operation of thymectomy for myasthenia gravis have been Blalock and Keynes. The disease which was described in 1895 by Jolly causes muscular weakness involving the extra-ocular muscles or those of the larynx and pharynx hand etc. It is subject to spontaneous remissions and exacerbations. There are many theories as to its causation one postulates the formation of a toxin with a curare-like action by the thymus. Neostigmine and edrophonium may be used for diagnosis as also may the anti-depolarizing relaxants. The operation for the relief of myasthenia gravis involves splitting of the sternum. One or both pleural cavities may be opened.

The mediastinum unless fixed by adhesions is deviated to the sound side and presses on the sound lung. If the lung is adherent to the chest wall these effects may not be marked. If the condition is not soon checked, death from cardiorespiratory depression follows.

Respiration is affected by —

- 1 Collapse of the lung and retraction of the mediastinum
- 2 PARADOXICAL BREATHING (Sometimes called internal paradoxical respiration to distinguish it from external paradoxical respiration seen in respiratory obstruction or in fourth plane anaesthesia when the chest and abdomen move paradoxically, the chest contracting during inspiration) — When lung tissue is unsupported by a rigid chest wall it tends to move in response to pressure changes in the bronchi which is high during expiration and lower during inspiration. These changes are minimal during quiet breathing but increase during deep or partially obstructed breathing. The partially collapsed lung on the affected side is emptied still more with each inspiration as air is drawn from it into the sound lung. During expiration as chest cavity gets smaller sound lung squeezes some of its vitiated air into the collapsed lung. (The remainder of its air enters the trachea.) Thus affected lung expands during expiration and collapses during inspiration and owing to the amount of vitiated air drawn from the affected lung into the sound one the latter cannot interchange gases efficiently.

The air passing from one lung to the other is sometimes called the pendulum air.

- 3 MEDIASTINAL FLAP — If the mediastinum is mobile and an open pneumothorax exists its structures tend to move across to the sound side drawn by the negative pressure there. effect is greater during inspiration than expiration. The result is pressure on the sound lung and interference with its efficiency. Respiration is stimulated both in rate and depth by the collapse of the lung and by hypoxia and hypercapnia and the hyperpnoea which follows this stimulation makes the mediastinal movement worse. If the mediastinum is fixed by inflammatory adhesions its movement is less marked nevertheless the sound lung has to take over the whole of the tidal exchange eventually causing muscular exhaustion and hypercapnia.

#### 4 OXYGEN LACK AND CARBON DIOXIDE EXCESS

Circulation is affected by —

- 1 ABSENCE OF NEGATIVE INTRATHORACIC PRESSURE which normally aids filling of the auricles the venous return and cardiac output are thus reduced
- 2 MEDIASTINAL FLAP due to respiration causing intermittent obstruction of the superior and inferior venae cavae and consequent tachycardia and hypotension
- 3 REFLEX DISTURBANCES AND CHANGES DUE TO POSTURE

The hazards of open pneumothorax are lessened if hyperpnoea is avoided. Measures to control them include — (1) Controlled breathing (2) The prevention of hyperpnoea consequent on atelectasis (3) Positive pressure anaesthesia

**B Minor Intrathoracic Operations —**

*Drainage of Empyema and Lung Abscess* —The risk in these cases is that the abscess should rupture into a bronchus and flood the bronchial tree. Therefore cough reflex should be retained and a regional block employed. The patient should be propped up or sitting or the bad side should be dependent (see Chapter XVII)

If general anæsthesia is used as in children or in the presence of sepsis of the chest wall induction must be smooth no coughing should be caused. Cyclopropane thiopentone or gas-oxygen-trilene are suitable and the head and diseased side should be kept low and a suction apparatus should be available

**C Major Intrathoracic Operations** —Pneumonectomies lobectomies removal of foreign bodies from thorax operation for cure of diaphragmatic hernia partial oesophagectomy ligation of patent ductus arteriosus transthoracic gastrectomy etc

**Special Problems.**—The patients are often in poor condition. The operation may be prolonged. Oozing and trauma may cause shock. Interchange of gases is often handicapped

Anæsthetic technique must —

- 1 Avoid toxic drugs
- 2 Prevent spread of sputum and blood through bronchial tree
- 3 Ensure adequate gaseous exchange. Hypoxia in thoracic as in other operations is a severe cardiac handicap
- 4 Prevent coughing during induction and maintenance of anæsthesia. Coughing with an open chest tends to spread infection and is itself inefficient as a means of clearing the airways of secretions
- 5 Ensure the return of the cough reflex at the end of operation
- 6 Prevent paradoxical breathing and mediastinal flap
- 7 Allow for aspiration of pus mucus etc

Secretions are troublesome as they (1) Cause respiratory obstruction (2) May spread infection. They are dealt with by (a) Suction (b) Posture—either the prone position with slight tilt or the lateral position with steep tilt (c) Isolation of the diseased area of lung by a cuffed suction catheter (blocker) or an endobronchial tube

**HAZARDS OF OPEN PNEUMOTHORAX**

Normally the lungs are kept inflated by —

- 1 The atmospheric pressure acting on the alveoli
- 2 The adhesion of the two layers of the pleura due to the surface tension of the thin layer of fluid separating them. When the chest is opened atmospheric pressure becomes equal on the alveolar and pleural surfaces and the elastic recoil causes collapse of the lung

When one side of the chest is opened, negative pressure is lost on both sides. The larger the hole the more pronounced the effect. The lung on the affected side collapses due to recoil of its elastic tissues

- 2 THE INFLATION OF HYPERINFLATION CONSEQUENT ON ATLECTASIS — The reflex can be broken on the afferent side by deep anaesthesia on the efferent side by the use of a muscle relaxant at the centre by respiratory depressants
- 3 CONTINUOUS POSITIVE PRESSURE ANAESTHESIA — Manual pressure applied to the reservoir bag increases intra bronchial pressure during both inspiration and expiration. It may be necessary either intermittently or almost constantly

In its favour are the following points —

- a It increases oxygenation and prevents any collapse in the sound lung
- b It prevents mediastinal flap
- c It enables reinflation to be made of collapsed region of lung during the operation
- d It facilitates reinflation of collapsed lung at end of operation. A pressure of 12 mm Hg should seldom be exceeded unless testing for bronchopleural fistula. If expansion requires great pressure the cause may be respiratory obstruction due to a kink in the tube or secretions or to the endotracheal tube slipping into a bronchus

Objections to it are —

- a The lungs are not properly emptied so that carbon dioxide excretion is deficient. Breathing is spontaneous so paradoxical respiration is not abolished
- b Respiration becomes forced during expiration
- c There is a risk of spread and impaction of secretions into smaller bronchi with production later of atelectasis
- d There is interference with the venous return to the heart
- e There is a rise in venous pressure and increased oozing

This method although much in vogue up to about 1937 is not used much to-day except just before the chest is closed to expand the collapsed lung. It is replaced by assisted or controlled breathing i.e. pressure on the bag during inspiration only.

The dangers of bronchopleural fistula are (a) Inadequate pulmonary ventilation due to escape of gases unless a flow rate of about 20 l a minute is used. (b) Entry of blood or pus into the respiratory tract requiring frequent suction and table tilting to aid drainage. Better still a bronchus blocker should be inserted beforehand.

#### RE EXPANSION OF THE LUNG AT THE END OF THE THORACOTOMY —

- 1 Without drainage. The chest wall is closed except for a small tube communicating with the pleural cavity. The lung is fully expanded by bag pressure the tube withdrawn and its small hole sutured.
- 2 A wide bore needle is inserted into the pleural cavity after its closure and connected to a sucker then pulled out when all the air is evacuated.
- 3 An intra pleural drainage tube is used and connected to an underwater seal.

Hazards of Open Pneumothorax *continued.*

✓ Coughing during thoracotomy tends to cause asphyxia

✓ **1. CONTROLLED RESPIRATION** (which may start off as assisted respiration) — By manual or mechanical pressure on the reservoir bag during inspiration only both paradoxical breathing and mediastinal flap are abolished. It is really intermittent positive pressure anæsthesia. Expiration should be allowed to take at least twice the time given to inspiration. In this way complete deflation of the chest with proper carbon-dioxide elimination is ensured while the number of heart beats occurring during the period of normal intrathoracic pressure will be greater than the number taking place during the phase of abnormally high intrathoracic pressure when the venous return is smaller than normal. Before the chest is opened the pressure needed to inflate the lungs is about 20-30 cm  $H_2O$ . After thoracotomy it is about 10 cm  $H_2O$  and during controlled respiration the intrabronchial pressure varies between atmospheric and + 10 cm  $H_2O$ . It is difficult to build up more than 20-30 cm water, using a thin rubber bag.

Apnoea may result from one or more of the following —

- ✓ (a) Reflex inhibition of the respiratory centre due to distension of the lungs
- ✓ (b) Apnoea
- ✓ (c) Central depression from drugs
- ✓ (d) Peripheral muscular paralysis by a relaxant (the usual method in use to-day)

The advantages of controlled breathing are —

- ✓ 1 Paradoxical breathing and hypoventilation are corrected
- ✓ 2 Mediastinal flap is abolished
- ✓ 3 Control of the operative field is facilitated and movement can be made to suit the surgeon
- ✓ 4 Work done by the patient is reduced
- ✓ 5 The feel of the reservoir bag aids the anaesthetist in his assessment of anaesthetic depth

During intrathoracic surgery most anaesthetists in Britain employ controlled breathing from the start of the operation. The occasional use of a negative phase has some advantages and is provided by the newer machines for artificial ventilation. Before the chest is open a negative phase increases the venous return to the heart and is specially beneficial when the circulating blood volume is low. After the chest is opened a negative phase does not benefit the circulation significantly.

Mechanical respirators have been devised by Frenchner Moersch, Blease, James, Esplen, Beaver, Williams, Bang, Pask, Ritchie, Russell, Mortimer and others for use during anaesthesia.

The disadvantages of controlled respiration are —

- ✓ 1 Risk of rupture of emphysematous bulbar
  - ✓ 2 By reversing the intrathoracic pressures during the respiratory cycle the venous return is hindered
  - ✓ 3 Absence of respiration as a guide to anaesthetic depth
  - ✓ 4 Possibility of production of alkalosis from hyperventilation
- Probably of little clinical importance

quinidine lactate 100 mg intramuscularly can be substituted for this last dose. The intravenous injection of 100 mg in 1 per cent solution guardedly repeated may be used during operation. It may cause hypotension. The same drug may be given six hourly for the first two or three days post-operatively.

In recent years intravenous procaine has been extensively used during chest surgery. The average amount given is about 30 mg per minute. In children 3 mg per stone per minute can be given. It increases the threshold of cardiac irritability to stimuli, acts as an analgesic thus reducing the need for pethidine and thiopentone, it reduces the amount of broncho-spasm and it relaxes the veins so that the drip runs well. The chief sign of overdosage is hypotension. It is specially useful in heart surgery.\* At the present time however its use would appear to be declining†

Inotracheal intubation with a cuffed tube is usually desirable in major thoracic surgery. The oral method allows the use of larger tubes and facilitates suction. It can be performed under topical analgesia or following general anaesthesia with a relaxant e.g. thiopentone 0.2 to 0.6 g with *d* tubocurarine 15-30 mg or suxamethonium 50-100 mg. A combination of local and general can be used especially if endobronchial manipulations are to be carried out.

The patient's position on the table is usually the lateral one with a support at the level of the xiphisternum and another behind the sacrum. The hip and knees are strapped to the table to provide added support. A rise in carbon dioxide tension is very apt to follow any operation in the lateral position whether or not the pleura is opened in the absence of assisted or controlled breathing.

The prone position (Overholt-Larry Brown) is useful in wet cases as secretions will run into the trachea with a smaller tilt on the table than is required for this to be safely accomplished with the patient in the lateral position. Useful in upper lobectomies and in children too small for endobronchial blocking.

Before operation a drip should be set up, postural drainage should clear the bronchial tree as much as is possible, pre-operative bronchoscopy may be desirable in very moist cases.

Adequate physiotherapy and antibiotics are most necessary before operation.

### The Control of Secretions during Intrathoracic Operations :

Since the introduction of antibiotics and postural drainage the patient with excessive secretion is becoming uncommon but wet cases still occur in bronchiectasis, lung abscess, bronchopleural fistula and in degenerating new growths. Methods for preventing the spread of secretions include

\* Lar Brown, A I, d Sellik B A, *Anaesthesia* 1953 8 4

† — — — *Brit Med J* 1955 11 74

‡ McNiff R, *Proc World C* 5 of *Anaesthesia* 1956 55, M ne pols B g



## ANÆSTHETIC AGENTS

*Local analgesia* is used for bronchoscopy and for rendering the larynx insensitive. It commonly precedes a major thoracotomy either as a topical spray or as an injection through the cricothyroid membrane or trachea. Intubation may be carried out under local analgesia in patients with much sputum. Blockage of the vagus reflexes at the hilum of the lung with a long acting drug is often desirable. Block of the phrenic nerve in the thorax was at one time required to prevent excessive diaphragmatic movement but this is now controlled by paralysing the diaphragm with a muscle relaxant.

The combination of *nitrous oxide*, *oxygen*, *thiopentone* and a *muscle relaxant* is much in vogue at the present time. It is non explosive, does not demand a leak proof circuit, can supply plenty of oxygen, will control respiration easily and prevent coughing and bucking and similar reflexes. It may be used in a closed circuit or in a semiclosed one. *Intravenous pethidine* can be employed to supplement these agents and this reduces the amount of thiopentone injected and in addition relaxes the bronchi, encourages quick recovery of reflexes after anaesthesia and avoids the need for early post operative sedatives. A dose of 25-50 mg. is given as soon as the patient loses consciousness from thiopentone and additions of 10-20 mg. are given as required. The aim of this polypharmacy is to maintain respiratory depression, prevent movement, control reflexes and anaesthetize the patient. Returning bronchial reflexes and increased difficulty in inflating the lungs show that more relaxant is needed. A rising pulse rate, slight movements of a finger or limb or the face call for more sedative. While pethidine subdues bronchial reflexes, thiopentone controls muscular movement. Hyperventilation which results in hypocapnia should always play a big part in maintaining respiratory depression as its effects are rapidly reversible. Full respiratory activity must be present at the end of these operations.

*Cyclopropane* is considered by some workers to be the best anaesthetic for chest surgery. It is relatively non toxic, non irritating, rapidly recovered from, easy to produce, controlled respiration with, but it is flammable and causes cardiac arrhythmias.

*Ether* and oxygen still has its supporters. With ether controlled breathing is more difficult to produce and the respiratory centre must be depressed with morphine  $\frac{1}{4}$ - $\frac{1}{2}$  gr. intravenously in addition to the usual premedication. Recovery is thus retarded. Ether also has the reputation of irritating the bronchi but if the concentration of the vapour is increased gradually this is probably not marked.

If diathermy is employed in the chest *chloroform*, *trilene*, *halothane*, *pethidine* or *thiopentone* can be used temporarily if an inflammatory anaesthetic is being used.

To reduce cardiac irritability during intrathoracic surgery, some anaesthetists give *quinidine sulphate* 200 mg. by mouth the evening before operation and again one and a half hours before

quinidine lactate 100 mg intramuscularly can be substituted for this last dose. The intravenous injection of 100 mg in 1 per cent solution guardedly repeated may be used during operation. It may cause hypotension. The same drug may be given six hourly for the first two or three days post-operatively.

In recent years intravenous procaine has been extensively used during chest surgery. The average amount given is about 30 mg per minute. In children 3 mg per stone per minute can be given. It increases the threshold of cardiac irritability to stimuli, acts as an analgesic, thus reducing the need for pethidine and thiopentone; it reduces the amount of broncho-spasm, and it relaxes the veins so that the drip runs well. The chief sign of overdosage is hypotension. It is specially useful in heart surgery. At the present time however its use would appear to be declining.

Endotracheal intubation with a cuffed tube is usually desirable in major thoracic surgery. The oral method allows the use of larger tubes and facilitates suction. It can be performed under topical analgesia or following general anaesthesia with a relaxant, e.g. thiopentone 0.2 to 0.6 g, with *d* tubocurarine 15-30 mg or suxamethonium 50-100 mg. A combination of local and general can be used, especially if endobronchial manipulations are to be carried out.

The patient's position on the table is usually the lateral one with a support at the level of the xiphisternum and another behind the sacrum. The hip and knees are strapped to the table to provide added support. A rise in carbon dioxide tension is very apt to follow any operation in the lateral position, whether or not the pleura is opened in the absence of assisted or controlled breathing.

The prone position (Overholt, Parry Brown) is useful in wet cases as secretions will run into the trachea with a smaller tilt on the table than is required for this to be safely accomplished with the patient in the lateral position. Useful in upper lobectomies and in children too small for endobronchial blocking.

Before operation a drip should be set up, postural drainage should clear the bronchial tree as much as is possible, pre-operative bronchoscopy may be desirable in very moist cases.

Adequate physiotherapy and antibiotics are most necessary before operation.

### The Control of Secretions during Intrathoracic Operations †

Since the introduction of antibiotics and postural drainage the patient with excessive secretion is becoming uncommon, but wet cases still occur in bronchiectasis, lung abscess, bronchopleural fistula, and in degenerating new growths. Methods for preventing the spread of secretions include:

† Parry Brown, A. I. and Slack, B. A. *Anaesthesia* 1953, 8, 4.

† — — — *British Medical Journal* 1955, 71, 174.

† M. Field, R. Loe, H. McCullough *et al.* *Journal of Anaesthesia* 1956, 55, 101. Burg, P. *British Medical Journal* 1956, 2, 101.

*Anæsthetic Agents Control of Secretions continued*

- 1 Pre operative preparation postural drainage and antibiotic therapy
- 2 Regional analgesia during which the cough reflex is not lost—in thoracoplasty empyæma etc
- 3 Posture during operation When the patient is tilted 45° head down and is on his side secretions from the upper—diseased lung—will flow by gravity into the trachea and can be aspirated thus preventing contamination of the healthy lung Similarly if the patient is prone secretions can be aspirated (Overholt Parry Brown) Useful position in children too small for the use of blockers who are undergoing lobectomy and in upper lobectomies in adults When the patient is in the prone position the table should be tilted head down by 10° Anæsthesia in the prone position keeps the weight of the mediastinum off the sound lung and nearly always does away with the need for balloons except in very wet cases to prevent pus from the lower lobe contaminating the upper lobe In empyæma is often drained with the patient in the sitting position
- 4 Endobronchial intubation and blocking with inflatable cuffs

**ANÆSTHESIA FOR PNEUMONECTOMY**

**One lung Anæsthesia**—A one lung anæsthesia has the following advantages—

- 1 Blood and sputum can be confined to the diseased lung
- 2 The diseased lung is quiet and collapsed
- 3 Ventilation is not affected by a bronchopleural fistula
- 4 It reduces the need for aspirations and so saves interrupting a smooth anæsthesia
- 5 Surgeon can aspirate the open bronchus before closure and need not use clamps thus chance of subsequent bronchopleural fistula is reduced

The chief contra indications to selective intubation are lack of experience of the anæsthetist and smallness of the airways especially in the case of children Not all anæsthetists however favour one lung anæsthesia

One lung anæsthesia is accomplished by (a) Bronchial intubation (Gale and Waters 1931) of sound bronchus They passed a semi rigid cuffed tube via a laryngoscope into the trachea and thence blindly into the bronchus In 1936 Magill improved on this technique by threading his cuffed tube over a bronchoscope and placing it by direct vision in the selected bronchus (b) Bronchial tamponade of affected bronchus (Crafoord 1938) the anæsthetic being administered through an endotracheal tube (c) Bronchial occlusion with cuffed suction catheter (Magill 1934 and 1937 *Cern n Th mps n* 1943)

- 4 **BRONCHIAL INTUBATION**—A tube with inflated balloon is placed in the bronchus on the sound side

The normal relationships of the bronchi may be altered by disease Examination of the radiograph is often helpful Angle of deviation of left bronchus is about 45° from the vertical

of right bronchus 25. Left main bronchus is 5 cm long. Right main bronchus averages 4 cm in length before the upper lobe bronchus is given off while the upper lobe bronchus is at the level of the carina (Fig 59). Thus a tube in the right bronchus may block the orifice of the upper lobe bronchus with the production of hypoxia. Again a balloon inflated on a tube in the right main bronchus may extend into the trachea and occlude the left main bronchus preventing collapse of left lung and predisposing to poor

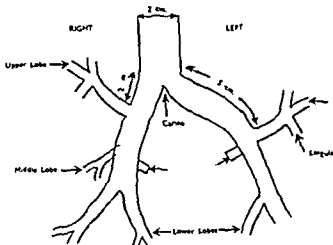


Fig 59.—Diagram of tracheobronchial tree

ventilation. It is thus important that placing of endobronchial tubes should be accurate. There are many experienced workers who regard the right main bronchus because of its shortness as being unsuitable for accommodating either an endobronchial tube or a cuffed suction catheter. The left main bronchus will readily accommodate either an endobronchial tube or a cuffed suction catheter.

1. **BLIND INTUBATION**—Very difficult on left side easier on right side. In recent years several new tubes (Stuertzbecher, Carlens, Macintosh and Leatherdale, Cren and Gordon etc.) have been developed. Concavity of tube should be towards side of bronchus to be intubated. If pushed too far right upper lobe bronchus may be occluded. If not far enough left main bronchus may be occluded. Careful auscultation of the lung zones will check air entry. A Magill tube size 6 to 10 is suitable.
2. **VISUAL INTUBATION WITH A CANNULA**—A long illuminated cannula is passed inside a cuffed armoured tube (Magill). Not often used to-day.
3. **VISUAL INTUBATION WITH A BRONCHOSCOPE**—The bronchoscope (8 mm) is passed inside a cuffed armoured tube (Magill).

*Anæsthetic Agents Control of Secretions continued*

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- 2 Regional analgesia during which the cough reflex is not lost—in thoracoplasty empyæma etc
- 3 Posture during operation When the patient is tilted 45° head down and is on his side secretions from the upper—diseased lung—will flow by gravity into the trachea and can be aspirated thus preventing contamination of the healthy lung Similarly if the patient is prone secretions can be aspirated (Overholt Parry Brown) Useful position in children too small for the use of blockers who are undergoing lobectomy and in upper lobectomies in adults When the patient is in the prone position the table should be tilted head down by 10° Anæsthesia in the prone position keeps the weight of the mediastinum off the sound lung and nearly always does away with the need for balloons except in very wet cases to prevent pus from the lower lobe contaminating the upper lobe An empyæma is often drained with the patient in the sitting position
- 4 Endobronchial intubation and blocking with inflatable cuff

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**A. BRONCHIAL INTUBATION**—A tube with inflated balloon is placed in the bronchus on the sound side

The normal relationships of the bronchus may be altered by disease Examination of the radiograph is often helpful Angle of deviation of left bronchus is about 45° from the vertical

is contemplated. In very most cases the tube can be placed before the induction of general anaesthesia.

The air passages must be freed of secretion by postural drainage before operation and by suction if necessary—before and also after the operation. The patient should breathe oxygen from a mask in the immediate post-operative period.

**B BRONCHIAL TAMPONAGE**—Blocking of the bronchus of the affected side followed by endotracheal anaesthesia.

After topical anaesthesia a bronchoscope is passed and  $\frac{1}{4}$  in. gauze moist with amethocaine is packed into the bronchus with a

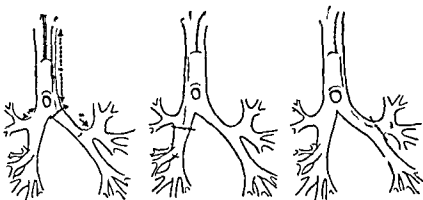


Fig. 61—McGill cuffed endotracheal tube in place.

packing probe. The gauze is kept in position by a long metal rod. Sputum is sucked out before the introduction of the gauze.

This will prevent collapse of the lung so that half the blood in the pulmonary circulation is prevented from interchanging its gases hypoxia and hypercarbia resulting. A non functioning lung is thus better collapsed and bronchial tamponade is not a good technique. Seldom used now.

**C BRONCHIAL OCCLUSION WITH CUFFED SUCTION CATHETER**—

A bronchus blocker is used e.g. that described by Vernon Thompson (Fig. 6). It is a soft rubber catheter of adequate length provided with a metal stylet for introduction through a bronchoscope (11 mm.) and a nylon covered rubber balloon which is inflated by the injection of 3 ml. of water. The blocker is accurately placed under local or general anaesthesia by bronchoscopic vision in the main bronchus of the diseased lung (or in the lower lobe bronchus for a lower lobectomy) the balloon inflated the stylet removed and the bronchoscope withdrawn. When the thorax is opened suction will collapse the lung. Anaesthesia is maintained by a cuffed endotracheal tube lying alongside the bronchus blocker placed via a laryngoscope.

One lung Anæsthesia—Visual Intubation *continued*

and afterwards withdrawn \*. The tube for the right bronchus has no rubber covering its distal inch of spiral wire—the spirals are also well separated to allow aeration of the right upper lobe bronchus. For left lung anæsthesia (right pneumonectomy) the endobronchial tube and balloon are in the left main bronchus without occluding the secondary bronchi—a physiological arrangement allowing the diseased right lung to collapse. For right lung anæsthesia (left pneumonectomy) the mouth

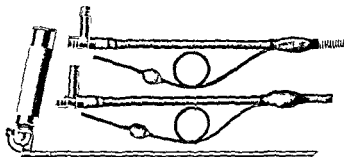


Fig. 63.—Magill's bronchoscope and right (with distal wire coil) and left endobronchial tubes. (Medical and Industrial Equipment Ltd.)

of the tube made of widely spaced coils of wire is in the right bronchus, part of the balloon being in that part of the main bronchus proximal to the upper lobe bronchus and part in the trachea. As this results in lack of collapse—or very slow collapse—of the left lung blood would continue until the pulmonary vessels have been ligated to circulate around the alveoli fail to become oxygenated and to part with its carbon dioxide. So for left pneumonectomy many workers choose not to use an endobronchial tube preferring an endotracheal tube with or without some form of bronchus blocker in the left main bronchus.

To sum up for left pneumonectomy an endotracheal tube with blocker in the left bronchus for right pneumonectomy an endobronchial tube in the left main bronchus or an endotracheal tube with blocker in the right main bronchus. Before these manipulations are carried out there should be adequate topical analgesia for bronchoscopy using posture to direct the analgesic solution into a bronchus if endobronchial manipulation

is contemplated. In very most cases the tube can be placed before the induction of general anaesthesia.

The air passages must be freed of secretion by postural drainage before operation and by suction if necessary—before and also after the operation. The patient should breathe oxygen from a mask in the immediate post-operative period.

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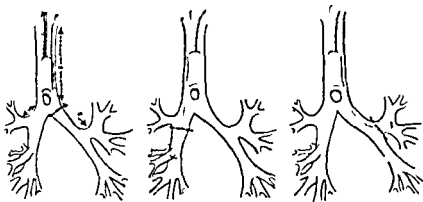


Fig. 61—Method of effecting endotracheal tube in place.

packing probe. The gauze is kept in position by a long metal rod. Sputum is sucked out before the introduction of the gauze.

This will prevent collapse of the lung so that half the blood in the pulmonary circulation is prevented from interchanging its gases, hypoxia and hypercarbia resulting. A non-functioning lung is thus better collapsed and bronchial tamponade is not a good technique. Seldom used now.

#### C BRONCHIAL OCCLUSION WITH CUFFED SUCTION CATHETER—

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**One lung Anæsthesia—Bronchial Occlusion continued**

The Magill blocker will go down an 8 mm bronchoscope. It can be used in large children and to occlude a secondary bronchus. It is smaller easier to place but more likely to slip than the Thompson instrument.

Bronchial blockers have the following advantages —

1. They are inserted into the lumen of the bronchus which is to be removed.
2. The airless lung or lobe gives room and tranquillity to the surgeon.
3. Drainage of the affected lung or lobe can be continued until the bronchus is clamped.



Fig 6 — Thompson bronchial blocker (Med. & Ind. Instr. Equipment Ltd.)

The disadvantages of endobronchial manipulation are —

1. Technical difficulty
2. Obstructing of upper lobe bronchus on the right side
3. Possibility of the tube or blocker slipping out of place
4. Possibility of rupture of bronchus or balloon

One lung anæsthesia is especially useful in cases of bronchopleural fistula, cyst, abscess and of course in pneumonectomy.

If one lung anæsthesia cannot be arranged, pneumonectomy can be performed under endotracheal anæsthesia (using a steep tilt or the prone position and suction if necessary to remove sputum).

In pneumonectomies, acute pulmonary oedema—the result of over transfusion—is a real risk, but post operative pleural effusion which is largely plasma usually represents 500 ml.

**ANÆSTHESIA FOR LOBECTOMY**

One lung anæsthesia is not desirable as it does not safeguard against spread of sputum into the healthy lobe of the affected lung. Secretion may be controlled by —

and in the trachea a small side channel is provided used either to aspirate secretions from the non-functioning lung or to distend it. (b) An inclined end bronchial cuffed suction blocker and end tracheal tube with inflatable cuff. For left lung surgery tube (b) is used. It is angulated so that it will blindly enter the left main bronchus. When the cuff on the blocker is inflated the left lung is isolated, respiration taking place through the right lung only. For right lung surgery the tube (a) is passed into the left main bronchus

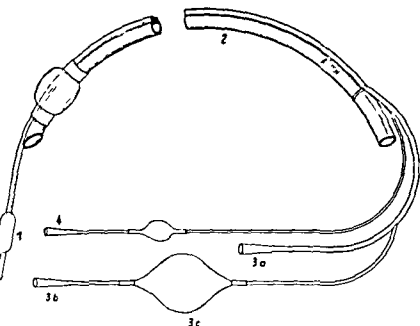


Fig. 11. The Sturtebacht combined endotracheal tube and bronchus blocker.

until it is stopped at the carina by its angulation. The endobronchial and if necessary the endotracheal cuff is now inflated while through the small side tube oxygen can be supplied to the right lung or secretions sucked from it. In some cases a rubber director aids insertion.

5. THE GREEN GORDON TUBE.—For surgery on the left lung involving right lung anaesthesia the Green Gordon tube can be employed\*. This is a tube provided with two inflatable cuffs. The lower 4 cm. of the tube (Magill sizes 8 and 9) is angulated at  $15^{\circ}$  and on the angulated portion is a lateral slot 2 cm.  $\times$  3 cm. wide with an inflatable cuff attached

**One lung Anæsthesia—Bronchial Occlusion continued**

The Magill blocker will go down an 8 mm bronchoscope. It can be used in large children and to occlude a secondary bronchus. It is smaller, easier to place, but more likely to slip than the Thompson instrument.

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F 62—A. M. n. Thompson's bronchial blocker (Med. & Ind. Instr. Co. Ltd.)

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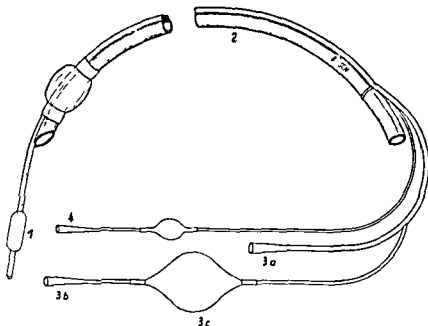


Fig 66—The tube blocker combined endobronchial tube and endotracheal blocker

until it is stopped at the carina by its angulation. The endobronchial and if necessary the endotracheal cuff is now inflated while through the small side tube oxygen can be supplied to the right lung or secretions sucked from it. In some cases a rubber director aids insertion.

- 5 THE GREEN GORDON TUBE—For surgery on the left lung involving right lung anaesthesia the Green Gordon tube can be employed\*. This is a tube provided with two inflatable cuffs. The lower 4 cm of the tube (Magill sizes 8 and 9) is angulated at 15° and on the angulated portion is a lateral slot 2 cm × 3 cm wide with an inflatable cuff attached

**One lung Anæsthesia—Bronchial Occlusion *continued***

The Magill blocker will go down an 8 mm bronchoscope. It can be used in large children and to occlude a secondary bronchus. It is smaller, easier to place but more likely to slip than the Thompson instrument.

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Fig 62 — Thompson bronchus blocker (Medical and Industrial Equipment Ltd.)

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One lung anaesthesia is especially useful in cases of bronchopleural fistula, cyst abscess and of course in pneumonectomy.

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In pneumonectomies acute pulmonary oedema—the result of overtransfusion—is a real risk but post-operative pleural effusion which is largely plasma usually represents 500 ml.

**FOR LOBECTOMY**

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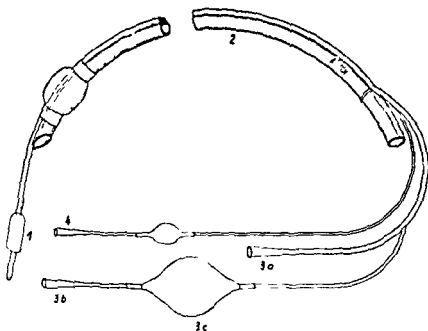


Fig. 11. The Stige tube (c) built into the left main bronchus blocker.

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Anæsthesia for Lobectomy—The Green Gordon Tube *continued*

to the margins of this aperture and surrounding the remainder of the tube. A stiff rubber hook projects from the tube opposite and 1 cm above the slot. The tracheal cuff has its lower margin 2 cm above the hook. A wire stilette is used during its insertion through the laryngoscope. It is passed

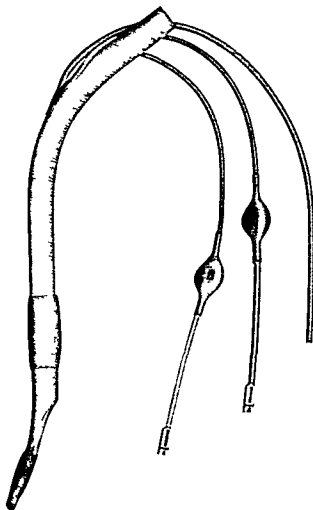


Fig. 67.—Miller bronchus blocker. (Medicine and Surgery, 1911, p. 111)

blindly into the right bronchus under topical or general anaesthesia through the mouth and comes to rest when its hook catches on the carina. The upper (tracheal) cuff is now inflated followed by the lower cuff in the right bronchus.

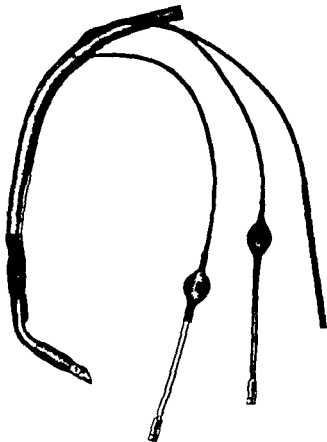


Fig. 18—Magill's bronchus tube (Medical and Industrial Equipment Ltd.)

The presence of breath sounds in the right lung and its upper lobe shows correct placing. In size the tubes correspond with Magill sizes 8 and 9.

Its advantages are—

- a It allows aeration of the whole of the right lung
- b It prevents contamination from the left lung
- c It prevents air leaks when the left main bronchus is cut
- d It aids in closure of the left main bronchial stump or of the tracheal wall



**anesthesia for Lobectomy—The Green Gordon Tube continued**

- e It is useful in high stenosis of the left main bronchus  
 neoplasm high up in the left main bronchus fistula  
 involving the left main bronchus plastic operations on  
 the left main bronchus For the majority of cases of  
 left lung surgery however an endotracheal tube alone  
 is satisfactory A blocker for the left or right upper lobe  
 bronchi has been described for use during resections of  
 the upper lobes

**ANÆSTHESIA FOR INTRATHORACIC NON PULMONARY OPERATIONS**

These include operations on the heart diaphragm œsophagus stomach mediastinum etc Endotracheal anæsthesia is all that is required and the same is true of almost all intrathoracic operations in children

Cardiac surgery often performed in children may involve division of a patent ductus arteriosus (Gross 1930) excision of aortic coarctation (Crafoord 1945) anastomosis of pulmonary artery to aorta or one of its main branches in tetralogy of Fallot (Blalock and Taussig of Baltimore 1945) or operation on the heart itself†

**Cardiolysis or Pericardectomy**—Thiopentone when given to a patient with constrictive pericarditis has a fearfully high mortality rate and should not be used Anæsthesia is induced with gas oxygen and ether or cyclopropane a relaxant is given and an endotracheal tube passed Maintenance is with the same anæsthetic or with pethidine given intravenously and with gas and oxygen.

**Anæsthesia for Mitral Valvulotomy‡** (and routine cardiac operations not requiring induced hypothermia) —The need here is for a perfect technique providing quiet smooth light anæsthesia without any hypoxia The special difficulties are associated with the open chest and with cardiac arrhythmia These patients have a relatively fixed cardiac output and do not tolerate deep anæsthesia *Premedication* Sedative drugs are helpful in all cardiac cases as they reduce oxygen demand Omnopon or morphine or nembutal are favoured in average or larger than average doses Children do well with omnopon gr — per stone Some give atropine or scopolamine others fear the tachycardia these drugs cause (which can even go on to acute pulmonary œdema) and omit them *Induction* is by thiopentone or by gas and oxygen or gas oxygen and ether It is the most critical stage of anæsthesia and strain in spasm coughing and hypoxia must all be avoided Preliminary local analgesia of the larynx is helpful After a relaxant has been injected a large cuffed endotracheal tube is inserted In children the cuff is usually unnecessary *Maintenance* Deep

anesthesia is contra indicated. Thiopentone gas and oxygen ether-oxygen gas (60 per cent) oxygen (40 per cent) and ether-pethidine gas (60 per cent) oxygen (40 per cent) have all been successfully used with or without a procaine drip (0.2 to 1 per cent). Some workers use procaine routinely others regard it as a cardiac poison and only employ it if arrhythmia becomes severe. From the start of the operation or when the thorax is opened controlled or assisted breathing is employed. Secretions are aspirated as required blood is replaced as it is lost taking care not to give too much. Some workers always set up two drips one in the arm the other in the foot.

There are workers of experience who employ only nitrous oxide oxygen and a relaxant † with the idea that possibly such drugs as thiopentone may in some cases handicap the circulation. Pre-medication is e.g. promethazine 50 mg. and atropine gr 1's. The patient is given pure oxygen which in three minutes displaces most of the nitrogen from the lungs. Nitrous oxide eight and oxygen two litres a minute are now given and after two minutes—or less if delirium ensues—a dose of *d* tubocurarine is given sufficient to cause apnoea e.g. 25–30 mg. A large cuffed tube is now passed. Respiration is controlled using a Waters circuit and a flow rate of nitrous oxide two litres and oxygen one litre a minute. A machine is not used to respire the patient. At the end of the operation atropine 1.3 mg. and when that has caused tachycardia neostigmine 2.5 to 5 mg. are injected. For emergency use a sterile giving set with wide bore needle should be at hand for giving blood into the aorta should it be necessary. *Arrhythmia*. Causes include pre-existing disease hypoxia hypercapnia reflex disturbances during intubation surgical manipulation inadequate depth of anaesthesia or relaxation rib spreading sudden haemorrhage drugs e.g. cyclopropane trichlorethylene belladonna alkaloids pressor amines cerebral embolism. Hypothermia may also cause arrhythmia. Prevention and treatment include digitalization and quinidine before operation and procaine or procaine amide (100–500 mg. intravenously in divided doses). Neostigmine 0.5 to 0.5 mg. given intravenously may reverse persistent tachycardia and multifocal ventricular extrasystoles. It is usual to have the electrocardiogram running and sinus tachycardia premature contractions auricular fibrillation and nodal rhythm may be encountered. Cardiac standstill demands oxygen inflation cardiac massage (artificial circulation) and perhaps intracardiac calcium chloride calcium gluconate nor-adrenaline. An electrical defibrillator must be used to abolish entricular fibrillation.

It is wise in heart cases to be on the look out for severe pulmonary congestion and cardiac asthma which may develop into acute pulmonary oedema. The treatment of this includes (1) Tilting patient up with legs hanging down. (2) Giving oxygen perhaps under slight positive pressure during inspiration. (3)

Schott S. and Helmworth F. W. *Proc. World Congress of Anaesthetists* 1956 75.  
Miephel B. K. *Int. J. Anaesth.* 1957 12 129.  
† G. V. T. C. and R. D. G. J. *Br. J. Anaesth.* 1957 12 129.

### **Anæsthesia for Mitral Valvulotomy continued**

Application of venous tourniquets high on limbs to reduce venous return to heart (4) Slow intravenous injection of 250 mg. of aminophylline (5) Aspiration of secretions from bronchi (6) Withdrawal of 500 ml. of blood (7) Injection of morphine intravenously, slowly.

**Anæsthesia for Operations to Close the Ductus**—The ductus is a wide channel between the distal part of the aortic arch and the pulmonary artery in foetal life when it conveys blood from the right side of the heart to the aorta by passing the functionless lungs. When the lungs expand after birth the ductus gradually closes completely in four weeks being replaced by the ligamentum arteriosum. If closure does not occur blood flows from the high pressure aorta to the low pressure pulmonary artery a reversal of the direction from intra uterine life. The results are (1) Increased intrapulmonary pressure (2) Right ventricular hypertrophy to deal with it (3) Small volume of blood passing down aorta with low diastolic blood pressure and high pulse pressure—these pressures become normal when the ductus is tied (4) Hypertrophy of left ventricle.

Cyanosis is not marked unless other congenital abnormalities exist also. There is a loud systolic and diastolic murmur the former being more pronounced. Endocarditis frequently coexists and greatly adds to the risks of operation.

A slow drip should be set up as grave hæmorrhage may take place while ductus is being cleared. Tying the ductus results in increase in the peripheral blood volume so drip must be slow.

Light ether anæsthesia through an endotracheal tube with just enough muscle relaxant to abolish breathing or thiopentone pethidine gas oxygen and a relaxant can be used. Cyclopropane may cause tachycardia or arrhythmia but this can often be controlled by the intravenous injection of 10 ml. of 1 per cent procaine or 0.5 per cent procaine painted on the surface of the heart.

Optimum time for operation is 5-12 years of age. In its absence expectation of life is halved.

**Anæsthesia for Repair of Coarctation of Aorta**—Induced hypotension has a useful place in anæsthesia for this condition. It reduces blood loss from the enlarged vessels in the chest wall and makes the actual suturing of the aorta easier. The blood pressure should be rising again when the clamps are taken off the aorta otherwise at this stage circulatory collapse may occur. The technique of general anæsthesia should present few special problems and the generally employed method is thiopentone relaxant oxygen inflation insertion of a cuffed tube controlled respiration with nitrous oxide and oxygen.

**Anæsthesia for Repair of Oesophageal Atresia in the Newborn**—The first successful operation was performed in 1943 by Haight and Townley in the U.S. Before the infant is brought to the theatre a fine polythene tube should be inserted high into an internal saphenous vein and through it a drip of fifth normal

saline and 4·3 per cent dextrose given. Premedication should be atropine gr  $\frac{1}{12}$  (0·3 mg). One method of anæsthesia\* is to give thiopentone 2·5 to 5 mg per lb of body weight and *d* tubocurarine 1 mg per 5 lb into the drip. Then after aspiration of secretions and insufflation with oxygen a 00 Magill armoured endotracheal tube is gently inserted into the larynx. Anæsthesia is maintained with equal parts of nitrous oxide and oxygen with a gas flow of 3-4 litres a minute and either a small to and fro absorber or other suitable circuit. Respiration is controlled until the end of the operation when, if necessary neostigmine 0·03 mg/lb is injected and the atropine (gr  $\frac{1}{12}$ ) repeated. A fine rubber catheter can be passed through the nose into the upper end of the œsophagus to enable the surgeon to locate the œsophagus.

Other workers prefer ether and oxygen through an endotracheal tube while some use only oxygen and *d* tubocurarine controlling the breathing throughout. This last is justified on the ground that the neonate feels no pain and is spared toxic drugs. See also Chapter XVI.

The technique of anæsthesia for intrathoracic surgery is not yet standardized. For further reference the following papers are recommended. MAGILL I W (1936) *Amer J Surg* 34 450. NOSWORTHY M D (1941) *Proc R Soc Med* 34 479. BEECHER H K (1940) *J Thorac Surg* 10 202. ORTON R. H (1947) *Med J Aust* 2 255. MILLAR C JOAN *Proc R Soc Med* 1952 45 51.

See also *The Principles of Thoracic Anæsthesia* by W W Mushin and I Rendell Baker 1953 Oxford Blackwell Scientific Publications.

Good papers dealing with anæsthesia for cardiac operations include those by Parry Brown A I and Sellick B A *Brit med Bull* 1955 11 174. Sellick B A *Proc World Congress of Anæsthesiologists* 1956 58 Minneapolis Burgess Publishing Co. Preston F S *Brit J Anæsth* 1953 25 299. Brown W M and Reid J E *Anæsthesia* 1954 68. Anæsthesia for Aortic Reconstruction see Thornton H L *Brit med J* 1957 1 253. For Elective Cardiac Arrest during Cardiectomy see Hale B F and others *Anæsthesiology* 1957 18 378.

**Cardiac Catheterization and Angiocardiography in Congenital Heart Disease**—Differential cardiac catheterization may be a long examination whereas angiocardiography consisting of the injection of diodone through a wide bore needle into the venous system followed by a series of X rays takes a shorter time but may be more dangerous than catheterization. The two investigations may be done on the same patient. Cardiac catheterization was first performed by Forsmann—on himself—in 1929. For this he was awarded the Nobel Prize in 1956. The investigation is not free from risk. In most children below 12 years old general anæsthesia should be used to avoid movement and distress. Patients are frequently in poor shape. The examination entails passing a small catheter from the forearm vein into the right heart.

Cardiac Catheterization and Angiocardiography *continued*

and pulmonary artery under X ray control. Pressures and samples of blood for gas analyses are taken. Some workers now allow oxygen to be given during the investigation. *Premedication*. In young children pentobarbitone (0.5 gr per stone per gram) and atropine. In older children rectal thiopentone (1 gr per 50 lb) with atropine or bromethol (125 mg per kg) with atropine. A test dose of quimidine is given before the investigation and if no headache, nausea or tinnitus is caused a larger dose gr 3-5 may be given by mouth two hours before operation to prevent cardiac arrhythmias.

Similarly a test dose of diodone should be given a day or two beforehand. Absence of stomatitis makes serious toxicity unlikely.

*Anæsthesia*—The sedated patient is placed on the X ray table and thiopentone is injected and supplemented with pethidine until the patient is settled. If no vein is available induction should be by nitrous oxide and oxygen until the catheter is inserted by cutting down on a vein; this can thereafter be used for intermittent doses of thiopentone. Endotracheal intubation is seldom necessary but all facilities for urgent intubation must be available. Thorough local infiltration with analgesic solution helps to prevent venous spasm during catheterization of the vein. Another method is to employ basal narcosis with rectal bromethol (125 mg/kg) and to inject suxamethonium 5 mg/tone just before the dye. Respiration is maintained by oxygen insufflation\*. With other workers again† cyclopropane induction and ether maintenance are preferred in spite of slight risk of explosion; a closed circuit is set up after oral insertion of a cuffed tube. Depth must be adequate to prevent bronchospasm resulting from injection of the diodrast. Blood pressure readings must be continuous and all means of resuscitation must be to hand such as methedrine, an efficient sucker etc. After the examination is completed the patient should remain under observation for fifteen minutes.

For angiocardiography it is usual to induce with thiopentone and maintain anæsthesia with nitrous oxide, oxygen and minimal trilene. No endotracheal tube is passed while respiratory reflexes associated with the injection of the contrast medium are controlled by small intermittent doses of thiopentone. Other workers employ basal narcosis with rectal bromethol (125 mg/kg) supplemented by small doses (0.5 mg/stone) of pethidine after the insertion of the intra-venous catheter. Yet another method is to give thiopentone and suxamethonium and to insufflate with oxygen. A pause in controlled breathing takes place while the contrast medium is injected and is then restarted until spontaneous respiration returns\*.

Duncalf D and Thompson P W. *Brit J Anaesth* 1957 28 450  
 \*Carnegie D M. *Brit Med J* 1951 77 230  
 †Baran D A. *N. Anaesthesia* 1953 8 131

## CHAPTER VIII

## THE COMPLICATIONS AND SEQUELÆ OF ANÆSTHESIA

## PULMONARY COMPLICATIONS

Abdominal operations adversely affect mechanical lung function by reducing total lung capacity and maximal inspiratory and expiratory flow rates due to mechanical obstruction bronchospasm and spasm of the muscles of the anterior abdominal wall secondary to pain

These may be expected to occur in 5 per cent of all operations in 10 per cent of abdominal operations

In 1900 Mikulicz showed that over a four year period chest complications followed general and local anæsthesia with equal frequency

**Pathology**—The conditions arise in association with —

- 1 Primary blocking of bronchi The most common
- 2 Primary interference with pulmonary blood supply
- 3 Primary bacterial invasion of lungs
- 4 Spread from disease in abdomen
- 5 Pneumothorax—rarely

The commonest complication is segmental atelectasis (Coryllos *J Amer med Ass* 1929 93 98) due to retention of sputum

- 1 PRIMARY BLOCKING OF BRONCHI AND RETENTION OF SPUTUM while blood supply remains intact
  - a Poor expulsive mechanism after operation due to (i) Pain (ii) Reduced movement of diaphragm (iii) Sedatives (iv) A tight binder or dressing (v) Spasm of muscles of abdominal wall The smaller bronchi are cleared by cilia and these may be inhibited by the anæsthetic used increased stickiness of sputum hypoxia hypercapnia etc
  - b Constriction of the bronchi Thus together with engorgement of the mucosa may (i) Follow reflexly from stimuli inflicted at too tight a plane of anæsthesia intubation traction on mesentery etc (ii) Result from post operative pain (iii) Be due to cholinergic drugs e.g. cyclopropane thiopentone neostigmine (iv) Be due to irritant anæsthetics (v) Be due to aspiration of stomach contents blood etc
  - c Excessive production of sputum due to (i) Smoking (ii) Infection etc

Mendelson† describes a post operative condition following the aspiration of acid irritating gastric contents characterized by asthma cyanosis dyspnoea and in severe cases acute pulmonary oedema

See Is Anscombe A R *Primary Complications of Abdominal Surgery* 1957 London

† Mendelson C L *Amer J Obstet Gynec* 1946 52 191

**Pulmonary Complications—Pathology continued**

- 2 **PRIMARY INTERFERENCE WITH PULMONARY BLOOD-SUPPLY—**
  - a Emboli—either blood clot or fat
  - b Acute pulmonary œdema Most common in hypertensive patients and in coronary disease May be brought on by too much intravenous fluid
- 3 **PRIMARY BACTERIAL INVASION OF LUNGS—**
  - a From aspirated material
  - b From contaminated anæsthetic equipment e.g. tubes face-masks etc
- 4 **SPREAD FROM DISEASE IN ABDOMEN—**The lymphatics can readily transmit infection through the diaphragm from the abdomen to the thorax e.g. in cases of subphrenic abscess or peritonitis Empyema or suppurative pneumonitis may result perhaps with bronchial fistula Multiple small lung abscesses may be part of a general pyæmia following peritonitis or acute abdominal infection
- 5 **PNEUMOTHORAX—**May occur spontaneously May follow trauma to pleura in operation on kidney gall bladder or thyroid etc May follow intercostal block

**Factors influencing Chest Complications—**

**TYPE OF OPERATION—**Most common after upper abdominal operations especially long ones Fairly common following operations for hernia Of non abdominal operations thyroid ectomy is most often followed by chest complications Pulmonary embolism commonest after pelvic operations

After laparotomy there is reflex fixation of the diaphragm and of the muscles of the anterior abdominal wall while reflex bronchospasm may also occur

The situation of the incision is important and if this can avoid the anterior abdominal wall so much the better (e.g. Mayo's incision for renal surgery a transthoracic incision for high gastrectomy etc.)

**Relationship to Oral Sepsis—**Pulmonary suppuration may occur in patients with dental sepsis (1) Independently of operation (2) Following operation under general anæsthesia (3) Following dental extraction Resulting pathology may be (a) suppurative bronchitis (b) suppurative pneumonitis (c) lung abscess (d) empyema Pulmonary abscess is very rare in edentulous patients The onset may be delayed for three weeks after operation As a routine dental sepsis should be removed three weeks before operation In patients undergoing major dental operation a careful pre-operative oral toilet adequate packing of the pharynx head-down position avoidance of drugs depressing the laryngeal reflexes and antibiotics are all useful preventive measures If inhalation of a foreign body is suspected early radiography and bronchoscopy are necessary

**SEX—**Males affected three times as frequently as females

**AGE—**More frequent in older age groups

**SEASON—**More frequent in cold weather

**SMOKING**—By causing bronchial catarrh smoking greatly increases incidence of chest complications

**ANÆSTHETIC**—Very little to choose between different agents and techniques if administration is skilful. The longer the duration of deep anæsthesia the greater will be the incidence of chest complications

#### POSSIBLE CAUSES—

- 1 Operation and anæsthesia reduce and inhibit ciliary action and lower resistance to endogenous and exogenous infection the latter either travelling via the air passages or through the lymphatics of the diaphragm from infected areas in the abdomen
- 2 Hypoventilation during and immediately after operation due to —
  - a Injudicious use of muscle relaxants
  - b Prolonged deep general anæsthesia
  - c High intradural or extradural analgesia with intercostal paralysis
  - d Pre-operative and post-operative sedative drugs which depress respiration e.g. morphine and bromethol
  - e Pain from operation site preventing adequate breathing and coughing. This was recognized as long ago as 1900 by Mikulicz and in 1902 by Campiche of Lausanne
  - f Reflex inhibition of diaphragmatic or thoracic movement associated with abdominal operation
  - g Awkward position on operation table as in gall bladder and kidney operations lithotomy and Trendelenburg positions
  - h Tight bandages
 Tidal exchange may be decreased by 50 per cent after upper abdominal operation. When the patient is in the lateral position bronchspirometry has shown that the ventilation of the lower lung is greater than that of the uppermost lung
- 3 Rapid absorption of gases from alveoli and the absence there of sustaining gases e.g. nitrogen. When cyclopropane and oxygen are given air should be introduced into the circuit towards the end of the operation
- 4 Intrabronchial aspiration of foreign material
- 5 Prolonged shock
- 6 Septic emboli from site of operation in infected cases
- 7 Atelectasis may be part of a reflex respiratory spasm the result of stimuli applied at too light a plane of anæsthesia

**Clinical Types.**—May vary from mild bronchitis to massive collapse but segmental atelectasis is the commonest finding. It is likely to be basal unilateral with onset sometime during the first forty eight hours. Progressing insidiously it may progress until there is malaise pyrexia slight cyanosis together with the signs of consolidation. The differential diagnosis is from (a) Lobar pneumonia (b) Pulmonary embolism (c) Cardiac infarction (d) Pulmonary tuberculosis (e) A surgical complication (f) Temporary paralysis of the diaphragm sometimes seen after operation



**Pulmonary Complications—Clinical Types continued**

- 1 Bronchitis
- 2 Atelectasis
  - a Segmental
  - b Lobar
- 3 Bronchopneumonia
- 4 Pulmonary embolism

**Diagnosis**—Atelectasis need cause no symptoms but it is likely to lead to trouble if it is associated with bronchial obstruction infection or a persistent interference with circulation associated with an arteriovenous shunt through the area of collapse. Post operative atelectasis was described by Wm Gardner of Glasgow in 1850. Onset of symptoms in first 48 hours probably points to bronchitis or atelectasis occasionally to massive pulmonary embolism. Atelectasis should be suspected if in the immediate post operative period the temperature rises to 101 F if the pulse rate is greater than 100 per minute and the respiration rate more than 20 per minute—always providing that there is no surgical reason to account for these signs.

Onset several days after operation is probably due to secondary bronchopneumonia from aspiration etc especially if signs of a fulminating pneumonia develop in a patient suffering from such conditions as intestinal obstruction persistent vomiting etc.

Primary bronchopneumonia can occur without previous atelectasis. Primary lobar pneumonia can also occur. It is important not to overlook the possible coexistence of abdominal abnormality e.g. peritonitis which may cause chest symptoms.

Sudden onset in second week is probably due to pulmonary embolism.

**SIGNS AND SYMPTOMS OF ATELECTASIS —**

- 1 Rapid breathing—30–60 a minute
- 2 Rapid heart rate
- 3 Absence of pain on inspiration unless pleural surface of lung is involved
- 4 Dilatation of alae nasi and slight cyanosis
- 5 Restricted movements of affected side of chest
- 6 Diminution of breath sounds and perhaps decreased resonance is almost normal after abdominal operations. When the condition is established signs of consolidation are present. Rhonchi may be present. Râles rare
- 7 Lower lobes usually involved
- 8 Mediastinal displacement towards affected side in gross cases
- 9 X ray appearance may resemble that of bronchopneumonia. Elevation of one or other side of diaphragm very common after upper abdominal operations in absence of clinical atelectasis. In atelectasis there is contraction of lung tissue in bronchopneumonia swelling. Occasionally an unresolving chest complication after operation is tuberculous.

The complications of atelectasis are bronchopneumonia pleural effusion and bronchiectasis. Massive collapse causes pain in

the chest of sudden onset dyspnoea cyanosis fever tachycardia and mediastinal shift

#### SIGNS AND SYMPTOMS OF PULMONARY EMBOLISM—

The classical signs are dyspnoea pleural pain and hæmoptysis. This is more common in medical than in surgical wards especially in patients with heart disease. Massive embolism more frequent in old than in young patients and in septic cases and those having undergone long and difficult operations. Condition usually associated with venous thrombosis in legs or pelvis. Onset may coincide with getting up or straining at stool. Sudden onset of pain in chest from second to fourteenth day after operation. Usually during second week. May be hæmoptysis early diminution of breath sounds dullness and tachycardia. Signs of pleurisy present in 90 per cent of cases but blood coughed up in only 40 per cent. Jaundice due to hæmolysis of the blood in the infarct may be seen.

If the embolus is massive there is profound shock sweating pallor air hunger and anxiety. Death usually follows quickly but recoveries have been reported. Trendelenburg's operation of embolectomy has been done occasionally. Small emboli are often overlooked the patient complaining only of faintness tightness in chest duration short no physical signs. Non fatal attacks are three or four times as common as fatal attacks.

*Dia nosis*—Should be thought of when there is tachycardia hæmoptysis jaundice or unilateral effusion after operation. It is often mis-diagnosed as atelectasis or pneumonia. Sudden pain in the chest within three weeks of operation is due to embolism unless it can be proved otherwise. Radiology may show in severe cases raised diaphragm and consolidation of the lower zones. Benefit is said to have resulted from stellate ganglion block i.e. assuming the existence (which is doubtful) of neurogenic reflex vasoconstriction. The condition accounts for about 6 per cent of deaths associated with surgical operation.

*Treatment*—Sedation anti-coagulants and oxygen. If further emboli appear after the prothrombin time is prolonged ligation of a femoral vein or even of the inferior vena cava must be considered.

**SIGNS AND SYMPTOMS OF LUNG ABSCESS**—When foreign material is introduced into the trachea it gravitates into the dependent apex of the lower lobe with the patient lying supine and into the dependent upper lobe with the patient on his side. These are the commonest sites of abscess. Onset may be mild after a latent period of two to ten days simulating bronchitis or early bronchopneumonia. Cough dry and hacking appears together with wasting chills anorexia etc. A leucocytosis develops and the temperature starts to swing. Radiology in the early stages usually shows an area of consolidation. When the abscess erodes into a bronchus foul mucopurulent sputum is coughed up and a minority of patients achieve spontaneous cure. Those who do not require postural drainage suitable antibiotic treatment and perhaps operation. Occasionally a lung abscess ruptures into the pleura forming an

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operation 1 ml of 1 per cent isoprenaline solution is inhaled by mouth from a hand inhaler three times daily followed by fifteen to twenty minutes postural drainage during which time the basal regions of the chest wall are submitted to clapping and vibratory percussion during expiration. It is done with the patient in the prone and in each lateral position. This thrice-daily drill is continued until tipping produces no sputum during the immediate pre-operative period. The patients are tipped and given the inhalation just before the premedication is injected and on return to the ward are similarly dealt with as soon as conditions allow starting off with inhalation alone. Treatment is maintained for about five days or until no more sputum can be coughed up. *Levo-isoprenaline—isoelin—*is less toxic than ordinary isoprenaline. It is said to have ten times the bronchodilator action of adrenaline. Each ml contains 4.2 mg of active base.

Another method of easing cough and liquefying sputum is the inhalation after nebulization of *alveaire*—a detergent in sterile alkaline solution. It lowers the surface tension of the sputum and facilitates its removal by coughing. Each treatment should last about half an hour and can be repeated three or four times daily.\*

2. **AT OPERATION**—Avoid over sedation but use adequate atropine or scopolamine. Employ careful anaesthetic technique. See that the patient is as little depressed at the end of the operation as possible. It is however an interesting fact that chest complications do not appear to increase in patients who have prolonged post-operative sedation the result of the phenothiazine derivatives. Endeavour to remove the respiratory depressant action of muscle relaxants before the patient leaves the theatre. Suck upper air passages clear of secretions blood vomitus etc after operation using a bronchoscope if contamination has been gross. Post-operative bronchoscopy is not however without its dangers. It may cause hypoxia and consequent circulatory depression and should be preceded and accompanied by oxygen insufflation and an intravenous injection of atropine to damp down vagal reflexes. If the pleura has been opened during operation the lungs must be re-inflated as the chest is closed and the pleural cavity kept free of air and exudation in the post-operative period.
3. **POST OPERATIVE**—Avoid excessive sedation and prohibit use of atropine. Get the patient moving about in bed as early as possible. Encourage deep breathing and coughing at least once each hour. The nurse should be constantly rallying the patient to avoid his hypoventilation and hypostasis. Cough should be especially encouraged soon after a dose of morphine the abdomen can be held firm while the patient is coughing. Pot iod in adequate dosage i.e. 15 to 75 gr t.d.s. definitely liquefies viscous secretions and increases their amount and so

**Pulmonary Complications—Diagnosis continued**

empyema and of course a bronchopleural fistula. Such an empyema should be drained with the patient sitting not lying down. Local analgesia should be used. These measures should prevent the patient being drowned in his own pus.

The earliest sign is a patch of consolidation. Later a fluid level may be seen.

**Prevention of Chest Complications**—Causal factors should be avoided as much as possible.

1. **PRE OPERATIVE**—Pre operative investigation of pulmonary mechanical efficiency deserves more attention than it now receives. The vital spiogram is simple to perform, takes little time, can be done by a technician, and causes little discomfort to the patient. It gives information on three aspects of pulmonary function—

- a The vital capacity is related to the functional volume of the lungs
- b The maximal expiratory rate is related to the elasticity of the lungs and chest wall
- c The maximal inspiratory rate is related to the efficiency of the muscles of respiration\*

If the vital capacity is reduced to 500 ml or below extra care will be needed to prevent pulmonary complications. Nursing in an oxygen helium atmosphere might have a place in their management.

Avoid operations during acute infections of upper respiratory tract. See that chronic infections are treated. Avoid smoking for three weeks before operation. Have a physiotherapist go over with a patient the chest exercises necessary after operation so that maximal thoracic breathing is possible. Have teeth attended to and sinus infection cleared up. Penicillin should be given before operation to patients in whom the risk of pulmonary complications is likely to be high. 1 000 000 units should be injected and if a cough develops 250 000 units may be given six hourly. Inhalations of penicillin are also useful (60 000 units of calcium penicillin in 2 ml of water). Encourage loss of weight before operation when necessary. Obese patients are more liable to post operative complications because of (1) Associated respiratory and circulatory disease (2) The reduced vital capacity in obesity (3) The poor tone of the fat laden respiratory muscles (4) The increased technical difficulty of the operation.

Palmer and Sellick† advise a routine which in their hands definitely reduces the incidence of atelectasis. It consists of the inhalation of isoprenaline (neoprenaline, aludrine, isupren, neodrenal) and postural drainage with vibratory and clapping percussion to the chest wall before and after

the patient in such a position that an area of collapse is above the bronchus supplying it. Inhalation of alevaure is useful in treatment as well as in prevention

Good results have followed the intravenous injection of 5 ml of nikethamide (preceded if necessary by 0.1 to 0.2 g of intravenous thiopentone). The explosive cough produced may clear the air passages. Intravenous injection of 2 ml of paraldehyde has a similar effect.

A few ml of penicillin solution can be injected into the trachea from the front of the neck through a fine needle. The irritation of the carina so produced may result in a useful cough.

Carbon dioxide and oxygen inhalations can be given and when hyperpnœa is maximal strong ether vapour can be added to produce coughing.

Intravenous procaine drops (0.1 per cent) have been advocated both to relieve pain and to dilate the bronchial tree. The analgesia unaccompanied by respiratory depression which they produce enables coughing and deep breathing to take place and lessens the need for sedatives of a depressant type.

The application of cocaine solution to the pyriform fossæ to paralyse the internal laryngeal nerves and aid bronchial dilatation has been recommended.

Should these treatments not be successful a Magill tube should be inserted into the trachea blindly if possible after topical analgesia of the larynx and a suction catheter introduced into the lower air passages. The coughing so produced is usually beneficial. In grave cases bronchoscopic suction should be carried out on one or more occasions using topical analgesia or internal laryngeal nerve block and intravenous atropine.

If the secretions seem to be imprisoned by bronchospasm the slow intravenous injection of 0.25 g of aminophyllin or cardiophyllin is helpful in relieving the bronchospasm.

The great danger of atelectasis is that it may be followed by bronchopneumonia or lung abscess.

## GASTRO INTESTINAL COMPLICATIONS

**Nausea and Vomiting.**—May be influenced by one or more of the following factors —

1. **ANÆSTHETIC AGENT AND TECHNIQUE**—Most likely to be produced by chloroform ether cyclopropane trlene and pethidine in that order. Thiopentone regional analgesia myoneural blocking agents and gas-oxygen in the absence of hypoxia are not so frequently followed by vomiting. Intradural and extradural block usually causes less vomiting than inhalation of a volatile anæsthetic. Hypoxia predisposes to vomiting. Duration of operation and depth of anæsthesia are unfavourable factors.
2. **TYPE OF PATIENT**—Some patients are ready vomiters e.g. in travelling after simple dietary indiscretions sufferers from bilious attacks. Suggestion and the example of surrounding patients are important factors. Suitable pre-operative reassurance and sales talk are important.

**Prevention of Chest Complications—Post-operative continued**

permits their expectoration. It should not be given for more than three or four days consecutively as a tolerance develops. Steam too decreases the viscosity of bronchial secretions but does not increase their amount.

Prevention of pulmonary embolism involves frequent post-operative movement of the legs, feet and toes. Post-operative thrombophlebitis is most frequent in old fat patients who have suffered from shock or hæmorrhage. Pre-existing varicose veins are a predisposing factor. The mutual adhesiveness of the blood platelets increases from the fourth to the twelfth post-operative day. Prolonged bed rest favours the condition. The possible sites of thrombosis should be examined frequently e.g. the calf, groins and feet. Homan's sign—pain in the calf on passive dorsiflexion of the foot indicates phlebothrombosis of calf and may demand ligation of the vein proximal to the clot. A low fever after a pelvic operation unless otherwise explained is probably due to thrombophlebitis which may be improved by lumbar paravertebral sympathetic block (L 2, L 3 ganglia). Should thrombophlebitis be suspected the blood prothrombin level should be lowered by tromexan or heparin. The intravenous injection of alpha tocopherol 300–600 mg daily has been recommended for the prevention of the condition.\*

Heparin 300 mg in one litre of saline given intravenously over 12 hours has an immediate effect (unlike tromexan which requires several days) and will prevent any further clotting. Protamin sulphate and blood transfusion antagonize the effects of heparin.

**Treatment —**

1. **BRONCHITIS** —Salpnaonamides, expectorants or sedatives as may be required. Inhalations of steam with menthol or tinct benzoin 1:10 are soothing in cases of tracheitis. Penicillin.

2. **ATELECTASIS** —The hourly shake up treatment —

a Turn patient in the bed

b See that he takes at least a dozen really deep breaths

c See that he coughs effectively if possible bringing up sputum

Good results have followed the injection into the lower seven intercostal nerves of long acting local analgesics e.g. procto-caine, efocaine, benzocaine 2 per cent with urethane 40 per cent in distilled water (Rappaport) and delatamin. The last named is ammonium sulphate and benzyl alcohol 0.75 per cent of each with sodium chloride 0.48 per cent in distilled water. It is claimed that the ammonium ion obliterates the C nerve fibre conduction leaving the motor fibres and skin sensation intact.

If the chest is forcibly knocked with the fist over the site of the collapse a plug of mucus may be dislodged from a bronchus during coughing. Postural drainage to prevent the patient coughing uphill may also be employed. It involves putting

\* Shultz, W. E. and Shute E. V. *Lancet* 1954, 1, 633

the patient in such a position that an area of collapse is above the bronchus supplying it. Inhalation of alevaire is useful in treatment as well as in prevention.

Good results have followed the intravenous injection of 5 ml of mikhethamide (preceded if necessary by 0.1 to 0.2 g of intravenous thiopentone). The explosive cough produced may clear the air passages. Intravenous injection of 2 ml of paraldehyde has a similar effect.

A few ml of penicillin solution can be injected into the trachea from the front of the neck through a fine needle. The irritation of the carina so produced may result in a useful cough.

Carbon dioxide and oxygen inhalations can be given and when hyperpnœa is maximal strong ether vapour can be added to produce coughing.

Intravenous procaine drips (0.1 per cent) have been advocated both to relieve pain and to dilate the bronchial tree. The analgesia unaccompanied by respiratory depression which they produce enables coughing and deep breathing to take place and lessens the need for sedatives of a depressant type.

The application of cocaine solution to the pyriform fossa to paralyse the internal laryngeal nerves and aid bronchial dilatation has been recommended.

Should these treatments not be successful a Magill tube should be inserted into the trachea blindly if possible after topical analgesia of the larynx and a suction catheter introduced into the lower air passages. The coughing so produced is usually beneficial. In grave cases bronchoscopic suction should be carried out on one or more occasions using topical analgesia or internal laryngeal nerve block and intravenous atropine.

If the secretions seem to be imprisoned by bronchospasm the slow intravenous injection of 0.5 g of aminophyllin or cardio-phyllin is helpful in relieving the bronchospasm.

The great danger of atelectasis is that it may be followed by bronchopneumonia or lung abscess.

### GASTRO INTESTINAL COMPLICATIONS

**Nausea and Vomiting.**—May be influenced by one or more of the following factors—

- 1 **ANÆSTHETIC AGENT AND TECHNIQUE**—Most likely to be produced by chloroform ether cyclopropane trlene and pethidine in that order. Thiopentone regional analgesia myoneural blocking agents and gas-oxygen in the absence of hypoxia are not so frequently followed by vomiting. Intradural and extradural block usually causes less vomiting than inhalation of a volatile anæsthetic. Hypoxia predisposes to vomiting. Duration of operation and depth of anaesthesia are unfavourable factors.
- 2 **TYPE OF PATIENT**—Some patients are ready vomiters e.g. in travelling after simple dietary indiscretions sufferers from bilious attacks. Suggestion and the example of surrounding patients are important factors. Suitable pre operative reassurance and sales talk are important.



**Prevention of Chest Complications—Post operative continued**

permits their expectoration. It should not be given for more than three or four days consecutively as a tolerance develops. Steam too decreases the viscosity of bronchial secretions but does not increase their amount.

Prevention of pulmonary embolism involves frequent post operative movement of the legs feet and toes. Post operative thrombophlebitis is most frequent in old fat patients who have suffered from shock or hæmorrhage. Pre-existing varicose veins are a predisposing factor. The mutual adhesiveness of the blood platelets increases from the fourth to the twelfth post-operative day. Prolonged bed rest favours the condition. The possible sites of thrombosis should be examined frequently e.g. the calf girth and feet. Homan's sign—pain in the calf on passive dorsiflexion of the foot indicates phlebothrombosis of calf and may demand ligation of the vein proximal to the clot. A low fever after a pelvic operation unless otherwise explained is probably due to thrombophlebitis which may be improved by lumbar paravertebral sympathetic block (L<sub>2</sub> & L<sub>3</sub> ganglia). Should thrombophlebitis be suspected the blood prothrombin level should be lowered by tromexan or heparin. The intravenous injection of alpha tocopherol 300–600 mg daily has been recommended for the prevention of the condition.\*

Heparin 300 mg in one litre of saline given intravenously over 12 hours has an immediate effect (unlike tromexan which requires several days) and will prevent any further clotting. Protamin sulphate and blood transfusion antagonize the effects of heparin.

**Treatment—**

1. **BRONCHITIS**—Sulphonamides expectorants or sedatives as may be required. Inhalations of steam with menthol or tinct benzoini co are soothing in cases of tracheitis. Penicillin.

2. **ATELECTASIS**—The hourly shake up treatment—

a Turn patient in the bed

b See that he takes at least a dozen really deep breaths

c See that he coughs effectively if possible bringing up sputum

Good results have followed the injection into the lower seven intercostal nerves of long acting local analgesics e.g. procto-caine efocaine benzocaine 2 per cent with urethane 40 per cent in distilled water (Rappaport) and dolamin. The last named is ammonium sulphate and benzyl alcohol 0.75 per cent of each with sodium chloride 0.48 per cent in distilled water. It is claimed that the ammonium ion obliterates the C nerve fibre conduction leaving the motor fibres and skin sensation intact.

If the chest is forcibly knocked with the fist over the site of the collapse a plug of mucus may be dislodged from a bronchus during coughing. Postural drainage to prevent the patient coughing uphill may also be employed. It involves putting

the patient in such a position that an area of collapse is above the bronchus supplying it. Inhalation of alveaire is useful in treatment as well as in prevention.

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Gastro-intestinal Complications—Nausea and Vomiting *continued*

- 3 **CONDITION OF STOMACH**—Vomiting is likely unless the stomach is empty. Reflex pylorospasm due to anxiety may delay gastric emptying. Swallowing of ether impregnated mucus or blood is a cause which can be lessened by suction from the mouth and pharynx after operation. Sipping of water soon after recovery of consciousness may cause vomiting. Post-operative thirst should be treated by mouth washes and rectal tap water.
- 4 **OPIATES**—Given before and after operation make some 30 per cent of patients vomit. Heroin is said to cause less vomiting although more respiratory depression than morphine. *Papaveretum is not superior to morphine in preventing nausea and vomiting.* Nembutal before operation and pethidine before or after operation may be substituted.
- 5 **TYPE OF OPERATION**—Vomiting is most frequent after laparotomy and especially after operation on the biliary tract. Dilatation of the cervix uteri often produces vomiting after operation.
- 6 **SEX**—More frequent in women than in men.

**TREATMENT**—This consists largely in preventing the causal factors whenever possible. Pyridoxine 100 mg before and once after operation is said to lessen the incidence of vomiting. Sod phenobarbiton 2 gr hypodermically may also be useful. The antihistamine drug dimenhydrinate (dramamine) given 50 mg before operation 50 mg immediately afterwards and then four hourly for four doses is said to reduce the rate of vomiting after operation by 50 per cent while avomine has also proved beneficial.

Cyclizine hydrochloride (marazine) 50 mg by deep subcutaneous injection half an hour before and several times after operation is said to be free from unpleasant side effects and to reduce vomiting\*. Promethazine 50 mg intramuscularly during operation and 25 mg post-operatively for four doses four hourly is said to reduce vomiting after operation most definitely†. Chlorpromazine will also reduce the incidence of vomiting and can be given before and after operation by mouth (25 mg the night before operation repeated before and immediately after operation) by injection and per rectum as a 300 mg suppository‡. Perphenazine (trilafon, fentazin) 5 mg given intramuscularly at the end of the operation reduces vomiting significantly. It is a derivative of phenothiazine§.

Glucose given for a day or two before operation may be helpful especially in children. A glucose (10 per cent) enema can be given immediately after the operation and can be combined with 10 units of insulin. A little old carbohydrate food given soon after recovery of consciousness is sometimes helpful.

Marcus P. S. and Sheehan, J. C. *Anesthesiology* 1955 18 423

† Gordon R. A. and others, *Canad. J. Anesth. Soc. J.* 1955 2 95

‡ Boulton, T. B. *Anesthesia* 1955 10 235

§ Scurr C. F. and Robbie D. S. *Brit. med. J.* 1958 1 922 and Moore D. C. and others, *Anesthesiology* 1958 18 72

Vomiting persisting for more than twenty four hours may be helped by —

- a The rapid swallowing of a pint of warm water containing a teaspoonful of sodium bicarbonate. This will probably be returned and will wash out the stomach with benefit
- b The intramuscular injection of dilute solution of chlorpromazine (12.5-25 mg)
- c The insertion of a Ryle's stomach tube through the nose with frequent gastric lavage and aspiration

**INTESTINAL DISTENSION** — Frequently follows laparotomy due to handling of intestine traction on mesentery and stimulation of autonomic ganglia. Other causes are air swallowing and partial inflation of the stomach from assisted or controlled respiration. Treatment (1) Heat to abdomen (2) Use of a rectal tube (3) Pitressin 10 units hypodermically it relaxes the small bowel while stimulating the colon (morphine has the opposite effect) (4) Neostigmine (0.5 mg) stimulates the small gut and inhibits the colon (5) Hypertonic sodium chloride 20 ml of 10 per cent solution intravenously (6) Enemata (7) Pantothenic acid part of the vitamin B complex given as the calcium salt 50 mg in 1 ml injected intramuscularly. This dose can be repeated

**Difficulty in Micturition** — Occurs more frequently after spinal than after other methods of anaesthesia. Occurs in about 10 per cent of cases following general anaesthesia but in these the anaesthetist is not held responsible. It is influenced by —

- 1 **TYPE OF PATIENT** — Most common in anxious apprehensive type. Patients with an enlarged prostate although managing well before operation may develop retention after operation
- 2 **TYPE OF OPERATION** — Most common after abdominal and pelvic operations including haemorrhoidectomy. A rectal tube predisposes to difficulty with micturition
- 3 **ATTITUDE OF SISTERS AND NURSES** — Incidence of condition differs from hospital to hospital. In some wards it is expected and so is frequently encountered
- 4 **DEEP SEDATION** — By removing desire for micturition deep sedation may allow the bladder walls to become stretched with subsequent difficulty in voiding

#### **TREATMENT —**

- 1 Encouragement and suggestion
- 2 Sit patient up in bed if possible with legs over side of bed
- 3 Drugs potassium acetate 60 gr two hourly for three doses. potassium acetate is both a diuretic and a parasympathetic stimulant. Carbachol pituitrin neostigmine etc
- 4 Catheterization should not be left too long otherwise stretching of bladder wall will occur making natural voiding more difficult

**Hiccup** — Persistent hiccup may follow anaesthesia either general or regional. It is a state of intermittent spasm of the diaphragm accompanied by sudden closure of the glottis. The central part of the diaphragm is supplied by the phrenic nerves the peripheral

**Gastro intestinal Complications—Hiccup continued**

parts by the lower six or seven intercostal nerves. The phrenic nerves contain motor sensory and sympathetic fibres the intercostals mostly sensory fibres.

**CAUSES**—Stimulation of sensory nerve endings of phrenic which are connected with the coeliac and other intra abdominal autonomic plexuses. The vagus may also act as part of the afferent arc of the reflex. Thus through these pathways hiccup may arise from impulses in any abdominal or thoracic viscus.

Central stimulation of the medulla may be causal in e.g. alcoholic intoxication uræmia encephalitis.

**TREATMENT—**

- 1 Periodic inhalation of carbon dioxide to produce hyperpnœa. Thus may be carried out for as long as it is beneficial.
- 2 Inhalation of amyl nitrite.
- 3 Deflation of the gut by neostigmine vasopressin etc.
- 4 Sedatives.
- 5 Intravenous fluid.
- 6 Injection of atropine.
- 7 Benzyl benzoate 2 ml. of 20 per cent solution by mouth two-hourly.
- 8 Quinidine 9 gr. by mouth or intramuscularly repeated two or three times at hourly intervals.
- 9 Muscle relaxants.
- 10 Hexamethonium salts.
- 11 Block of phrenic nerves—either unilateral or bilateral. 20 ml. of 1 per cent procaine or 1-1000 nupercaine or amethocaine with adrenaline is used. Injection is made at a depth of  $\frac{1}{2}$  in along a line extending 2 in. laterally from a point  $\frac{1}{2}$  in. above the sternoclavicular joint.
- 12 Division of phrenic nerves—either unilateral or bilateral. Radiology may determine which side of the diaphragm is at fault before division of the phrenic nerve in extreme cases. Unfortunately even bilateral division of the phrenic nerves may fail to cure hiccup because of the associated spasm of the intercostal and accessory respiratory muscles.

**MISCELLANEOUS****Air Embolism—****CAUSES—****1 SURGICAL—**

- a Operations involving injury to veins in the neck, thorax, breast and pelvis; operations on the brain and cord in the sitting position.
- b Operations on the heart.
- c Uterine curettage and insufflation.

**2 DIAGNOSTIC AND THERAPEUTIC INJECTION OF AIR INTO—**

- a Peritoneum.
- b Pleural cavity.
- c Large joints.

d Urinary Bladder

e Tissue spaces e.g. the perinephric area

### 3 SURGICAL INJURY

#### 4 ACCIDENTAL ENTRANCE OF AIR —

a During intravenous infusions

b During intra arterial transfusion under positive pressure

**SIGNS AND SYMPTOMS** — If air enters the veins in any quantity (100–150 ml) it will go to the right heart and lung causing an air lock obstruction in the pulmonary artery. This may result in a loud precordial murmur the so-called mill wheel murmur. There will also be sudden cyanosis hypotension tachycardia and hypopnoea followed by cardiac arrest.

**TREATMENT** — (1) Prevent further entrance of air into the circulation. (2) Lower the head end of the table to keep air out of the cerebral vessels. (3) Place patient on his left side so that bubbles are carried away from the mouth of the pulmonary artery. (4) If rapid improvement is not noticed the left thorax should be opened and manual cardiac compression carried out this will clear air out of the heart and great vessels. If air has been injected into an artery causing convulsions and rapid collapse or if it has passed through a congenital defect into the left heart manual systole will milk it away from the orifices of the coronary vessels. Defibrillation may be necessary.

Post mortem X rays of cases dying suddenly on the operating table might reveal this as a cause of death not infrequently.

**Encephalopathy** — A R Hunter† has described a type of encephalopathy following general anaesthesia characterized by light coma without definite localizing symptoms in the central nervous system some hours after apparent recovery from general anaesthesia. All of his cases have ended fatally and post mortem there is found cerebral congestion subarachnoid haemorrhage and areas of cerebral softening. It is not due to fat embolism cerebral thrombosis or hypoxia and is not associated exclusively with any particular anaesthetic or operation although several of the cases followed gas-oxygen and trilene anaesthesia for radical mastectomy. Seldon and his colleagues have reported similar cases from the Mayo Clinic ‡ one of them a radical mastectomy. They suggest that the cause is cerebral oedema and advise the intravenous injection of 100 ml doses of 25 per cent human serum albumin. Their cases completely recovered.

**Hyperpyrexia** — This may be seen after anaesthesia and may progress to convulsions and death §

**Post operative Paralysis of Upper Extremity** — First reported by Budinger in 1894. The most dangerous position is the Trendelenburg tilt with arm abducted to a right angle fully supinated and externally rotated. The condition formerly thought to be due to

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† Hunter A R *Lancet* 1949 1 1045

‡ Seldon, T H and others *Proc May Cl* 1949 24 14

§ Brown R L *Br Med J* 1954 1 15 6

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**Post-operative Paralysis of Upper Extremity continued**

pressure on nerves e.g. by shoulder braces is now thought to be secondary to stretching of nerves over the head of the humerus which the abducted position makes prominent. Additional factors are tying the arm or arms to an armboard behind the coronal plane of the body and full rotation of the head and neck toward the opposite side. Cases have occurred following the use of the gall bladder bridge. The upper part of the brachial plexus is more commonly involved than the lower part. Effects usually on motor nerves not sensory. To avoid stretching the plexus the following measures should be taken. (1) Shoulder braces must be padded and must make contact with acromion as far laterally as possible but they are better done away with the patient being supported on a non slip mattress (Langton Hewer\*). (2) Arm board must be built up with pads to prevent backward displacement of arm. (3) Hyperextension and external rotation of elbow must be avoided. (4) Intravenous injections should be given with the arm at the patient's side using an apparatus such as that described by Lee†. Prognosis is good but recovery may take months. The deltoid biceps and brachialis are the muscles usually affected.

**Perforation of Ear Drums ?**—This is due to intermittent positive pressure respiration compressing the air in the mouth and pharynx so that with widely dilated Eustachian tubes and a thin and atrophic tympanic membrane perforation may occur. Cleanliness and if necessary antibiotics form the correct treatment.

**Post-operative Anuria**—Due to —

- 1 PRERENAL —Fall in blood pressure
- 2 RENAL —Damage to renal tubules by (a) Anoxia (b) Toxins—
  - (i) Bacterial (ii) Products of tissue autolysis. Clinically acute glomerulonephritis, bilateral cortical nephrosis and acute tubular necrosis are described. The last may be an acute toxic nephrosis due to chemicals such as mercury or may be a lower nephron nephrosis due to hæmolytic sulphonamide sensitivity or shock.
- 3 POSTRENAL —Ureteral obstruction bladder neck obstruction etc.

If there is oliguria or anuria fluid should not be pushed and a continuous intragastric drip set up consisting of glucose 400 g, peanut oil 100 g and water 1 litre emulsified with acacia. This gives limited fluid and calories but neither protein nor salts. Spontaneous cure may come about during the second week of such treatment.

Blockage of the splanchnic nerves may be useful and may be accomplished by one of the following routes. (1) Sacral extradural (2) Lumbar extradural (3) Subarachnoid (4) Paravertebral. The block must ascend to the eighth thoracic nerve root.

\* Hewer C. L. *Anæsthesia* 1953 8 95  
 † Lee J. A. *J.A.A.* 1954 7 256.  
 ‡ Whittingham J. S. R. *Br. J. Med.* 1954 2 970.

The following conditions may be noticed after operation and anaesthesia. Reasonable care and common sense will prevent most of them —

Cutting of lips bruising of face and tongue excoriation of pharynx and larynx chipping or knocking out of teeth damage to eyes strain of muscles of back and limbs

Cardiovascular accidents may occur during or soon after anaesthesia while mental changes may be noticed afterwards especially in elderly patients. Abnormalities which are present before operation should be noticed so that operation and anaesthesia are not blamed for them

**Acute Adrenocortical Deficiency** — This usually shows itself during the first day or two after operation as acute circulatory collapse with hypotension tachycardia pallor and sometimes pyrexia and unconsciousness. The state of adrenocortical deficiency may arise in those patients who have been treated for some time with cortisone and from whom the drug has been suddenly withheld before operation. The treatment is intravenous hydrocortisone 100 mg given in a drip\*. Sometimes noradrenaline is required in addition

See also good article The Pre and Post-operative Care of Patients receiving Cortisone or Other Steroid Therapy by de Mowbray R R *Postgrad med J* 1957 **33** (32)

**The Body's Response to Injury** — During the first few days after injury or operation the following changes occur but normally require no treatment (1) Rise in temperature and pulse (2) Reduced urinary secretion due to antidiuretic hormone (3) Loss of nitrogen for the first three to seven days followed by a positive nitrogen balance (4) Potassium loss for two to five days then potassium retention (5) Sodium retention (6) Water retention (7) Loss of weight due to oxidation of fat (8) Reduction in eosinophils which is delayed by general anaesthesia (9) Increased excretion of steroid hormones (10) A temporary period of starvation and relative calorie deficiency (See Moore I D and Ball M R *The Metabolic Response to Surgery* 1952 Oxford Blackwell)

**Post operative Hypotension** † — Causes may be (1) Cardiovascular (2) Respiratory (3) Pharmacological (4) Neurogenic (5) Haematological (6) Endocrine (7) Postural

It may result in minor or major brain damage sudden heart failure renal damage vascular thrombosis or embolism and may be specially serious in elderly patients. The facilities of a post operative observation room should be more widely provided

Lundy J S *Aesthesiology* 1953 **14** 376

† Bibo R C M and Little D M *J Amer med Ass* 1957 **165** 1529

## CHAPTER XXV

## PRODUCTION OF ISCHÆMIA DURING OPERATIONS

## Bleeding during Anæsthesia —

## 1 ANÆSTHETIC CAUSES —

- a All general anæsthetics cause release of vasomotor tone at the periphery
- b Respiratory obstruction
- c Hypercapnia
- d Hypoventilation
- e Coughing during induction or maintenance
- f Resistance in anæsthetic circuit

## 2 NON ANÆSTHETIC CAUSES —

- a Pathological conditions causing increase in the bleeding time or in the coagulation time of the blood
- b Venous congestion secondary to posture heart disease or lung disease
- c Conditions causing a rise in the B M R
- d Operations involving vascular tissues e.g. muscle or gland
- e A rise in blood pressure

Ischæmia during operation has been produced by —

- 1 Total spinal analgesia (Griffiths H W I and Gilhes J *Anæsthesia* 1948 3 134) See p 290
  - 2 High extradural block (Bromage P R *Ibid* 1951 6 76) See p 306
  - 3 Ganglionic blocking agents (Enderby G E H and Armstrong Davison M H *Proc R Soc Med* 1951 44 829 Aserman D *Brit med J* 1953 2 061 Payne J P *Brit J Anæsth* 1953 25 134 Hampton L J and Little D M *Lancet* 1953 1 1299 Wyman J B *Proc R Soc Med* 1953 46 605 Enderby G E H *Proc World Congress of Anæsthesiologists* 1956 143 Minneapolis Burgess Publishing Co) See below
  - 4 Controlled arteriotomy (Bilsland W L *Anæsthesia* 1951 6 20) Used chiefly in neurosurgery but is losing popularity See p 445
  - 5 Application of negative pressure to the lower limbs after moderate hypotension has been induced by a ganglionic blocking agent (Saunders J W *Lancet* 1952 1 1786)
  - 6 Infiltration with adrenaline-saline 1-250 000
  - 7 Tourniquets during operations on limbs
- In the above groups 1 to 3 hypotension is normovolaemic with no reduction of circulating blood volume. In groups 4 and 5 hypotension is hypovolaemic as there is a reduction in the volume of circulating blood. The hypotension reduces the pressure in the great veins and reduces venous return and consequently

cardiac output. A fall in blood pressure results which in its turn by its effect on the carotid and aortic sinuses leads to tachycardia (Marey's law). Hypovolaemic hypotension is accompanied by intense vasoconstriction.

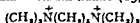
On the other hand total spinal and high extradural block result in the interruption of sympathetic vasoconstrictor impulses which travel through the anterior spinal roots in spinal block and through the anterior roots, mixed spinal nerves and white rami communicantes in extradural block. Hypotension does not depend solely however on the number of white rami or anterior roots blocked. This causes dilatation of the arterioles and pre-arteriolar capillaries so that the blood pressure falls. The effect is helped by reduction of the venous return and by paralysis of the sympathetic cardio-accelerator fibres resulting in further reduction of cardiac output. Hypotension here is accompanied by vasodilatation.

Ganglionic blocking agents produce their hypotension by increasing the capacity of the vascular bed relative to the volume of circulating fluid.

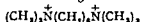
### GANGLIONIC BLOCKING AGENTS

Pentamethonium was first used as an antidote to the myoneural blocking effects of decamethonium by Organe, Paton and Zaimis in 1949 while in the same year M. H. Armstrong, Davison warned against its severe hypotensive action. Scurr in 1949 was the first deliberately to use the drug to lessen bleeding during surgery while Enderby and Wyman have been pioneers in its further use. Enderby stresses the need for posture to achieve satisfactory hypotension.

The formula of pentamethonium halide (C<sub>5</sub>) is



Hexamethonium has the following formula



The bromide of pentamethonium (lytensium) comes in 1 ml ampoules of 10 per cent solution and its iodide (antilusin) is put in ampoules of 2.5 ml containing 50 mg. Hexamethonium bromide (vegolysen) is sold in 10 ml ampoules of 1 per cent and 2.5 per cent and in 1 ml ampoules of 10 per cent solution and hexamethonium iodide (hexathide) in 2.5 ml ampoules containing 2 per cent solution. Hexamethonium tartrate is sold as vegolysin T and hexamethonium chloride as methium chloride, hexamethonium esormid and bistrum.

The hexamethonium salts are rather more constant in action than pentamethonium salts but even they fail to produce a dry operating field in 20 to 40 per cent of cases. The subsequent injection of procaine amide hydrochloride will frequently convert these failures into successes.

Penta and hexamethonium act by blocking the transmission of preganglionic stimuli to postganglionic nerves and act at all autonomic ganglia. All preganglionic nerves are cholinergic. Three types of

## CHAPTER XXV

## PRODUCTION OF ISCHÆMIA DURING OPERATIONS

## Bleeding during Anæsthesia —

## 1 ANÆSTHETIC CAUSES —

- a All general anæsthetics cause release of vasomotor tone at the periphery
- b Respiratory obstruction
- c Hypercapnia
- d Hypoventilation
- e Coughing during induction or maintenance
- f Resistance in anæsthetic circuit

## 2 NON-ANÆSTHETIC CAUSES —

- a Pathological conditions causing increase in the bleeding time or in the coagulation time of the blood
- b Venous congestion secondary to posture heart disease or lung disease
- c Conditions causing a rise in the B.M.R.
- d Operations involving vascular tissues e.g. muscle or gland
- e A rise in blood pressure

Ischæmia during operation has been produced by —

- 1 Total spinal analgesia (Griffiths H W I and Gillies J *Anæsthesia* 1948 **3** 134) See p 90
  - 2 High extradural block (Bromage P R *Ibid* 1951 **6** 26) See p 306
  - 3 Ganglionic blocking agents (Enderby G E H and Armstrong Davison M H *Proc R Soc Med* 1951 **44** 829 Aserman D *Brit med J* 1953 **1** 961 Payne J P *Brit J Anæsth* 1953 **25** 134 Hampton L J and Little D W *Lancet* 1953 **1** 1299 Wyman J B *Proc R Soc Med* 1953 **46** 605 Enderby G E H *Proc World Congress of Anæsthesiologists* 1956 143 Minneapolis Burgess Publishing Co) See below
  - 4 Controlled arteriotomy (Bilsland W L *Anæsthesia* 1951 **6** 20) Used chiefly in neurosurgery but is losing popularity See p 445
  - 5 Application of negative pressure to the lower limbs after moderate hypotension has been induced by a ganglionic blocking agent (Saunders J W *Lancet* 1952 **1** 1286)
  - 6 Infiltration with adrenaline-saline 1-250 000
  - 7 Tourniquets during operations on limbs
- In the above groups 1 to 3 hypotension is normovolæmic with no reduction of circulating blood volume. In groups 4 and 5 hypotension is hypovolæmic as there is a reduction in the volume of circulating blood. The hypotension reduces the pressure in the great veins and reduces venous return and consequently

cardiac output. A fall in blood pressure results which in its turn by its effect on the carotid and aortic sinuses leads to tachycardia (Marey's law). Hypovolaemic hypotension is accompanied by intense vasoconstriction.

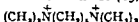
On the other hand total spinal and high extradural block result in the interruption of sympathetic vasoconstrictor impulses which travel through the anterior spinal roots in spinal block and through the anterior roots, mixed spinal nerves and white rami communicantes in extradural block. Hypotension does not depend solely however on the number of white rami or anterior roots blocked. This causes dilatation of the arterioles and pre-arteriolar capillaries so that the blood pressure falls. The effect is helped by reduction of the venous return and by paralysis of the sympathetic cardio-accelerator fibres resulting in further reduction of cardiac output. Hypotension here is accompanied by vasodilatation.

Ganglionic blocking agents produce their hypotension by increasing the capacity of the vascular bed relative to the volume of circulating fluid.

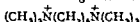
### GANGLIONIC BLOCKING AGENTS

Pentamethonium was first used as an antidote to the myoneural blocking effects of decamethonium by Organe Paton and Zaimis in 1949 while in the same year M. H. Armstrong Davison warned against its severe hypotensive action. Scurr in 1949 was the first deliberately to use the drug to lessen bleeding during surgery while Enderby and Wyman have been pioneers in its further use. Enderby stresses the need for posture to achieve satisfactory hypotension.

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Hexamethonium has the following formula



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Penta and hexamethonium act by blocking the transmission of preganglionic stimuli to postganglionic nerves and act at all autonomic ganglia. All preganglionic nerves are cholinergic. Three types of

**Ganglionic Blocking Agents** *continued*

blocking mechanism have been described \* (1) By depolarization of the ganglion cells an acetylcholine like action preceded by excitation e.g. by nicotine tetramethyl ammonium salts (2) By increasing the threshold for acetylcholine at the receptor cells—competitive inhibition the inhibition is not preceded by an excitatory stage—e.g. hexamethonium pendiomid pentolinium tetraethyl ammonium salts trimetaphan. This block can be reversed by sympathomimetic (or parasympathomimetic) drugs (3) By decrease in the amount of acetylcholine liberated by the postganglionic endings e.g. procaine and procaine amide thus procaine amide increases the ganglionic block produced by hexamethonium.

Hexamethonium thus acts by competitive inhibition of acetylcholine but is better not called an anticholinergic drug. Its action on the ganglia is analogous to that of *d* tubocurarine at the motor end plate.

**Pharmacology**—When given to the conscious patient hexamethonium causes postural hypotension fall in intra-ocular pressure dilated pupils (ciliary ganglion) and dryness of the eyes (sphenopalatine ganglion) dilatation of retinal and conjunctival vessels dry mouth and nose (otic and sphenopalatine ganglia and chorda tympani) dryness of the larynx (vagal ganglia) and skin reduction in gastric secretion and tone reduction in intestinal tone with constipation anorexia (enteric ganglia) and difficulty in micturition and impotence (pelvic ganglia). Fall in temperature relief of causalgia due to increase in blood supply. In diabetics there is a hypersensitivity to insulin through an effect on the suprarenal medulla so that a patient may go into hypoglycæmia without warning if otherwise normal doses of insulin are given. This may be shown during anaesthesia by progressive tachycardia.

During anaesthesia it causes (1) Low blood pressure or postural sensitivity of blood pressure (2) Release of autonomic tone by paralysis of ganglia at the preganglionic synapses of both sympathetic and parasympathetic systems the former predominating (3) Diminution of muscle tone (4) Disturbance of cardiac function (5) Respiratory depression (6) Dilated pupils (7) Reduction of operative shock.

Different ganglia are sensitive to hexamethonium in the following order—

- 1 Parasympathetic ganglia to salivary gland
- 2 Superior cervical ganglion
- 3 Vasomotor ganglia
- 4 Visceral ganglia.
- 5 Vagal ganglia in heart.

On the other hand hexamethonium causes no stimulation of nerve terminations or of muscle has no neuromuscular blocking effect has no atropine like action does not liberate histamine has no anticholinesterase activity. During hypotension vessels retain their sensitivity to adrenaline and the pressor amines.

Hypotension and decreased haemorrhage may be due to (1) The effects of gravity on blood distribution when the blood pressure is low (2) The effects of gravity on venous blood causing it to pool in the distended veins of the lower and dependent parts of the body (3) The reduced cardiac output consequent on the reduced venous return to the heart. As both sympathetic and parasympathetic ganglia are blocked hypotension depends on the original balance of these two. Where there is great parasympathetic tone its release will cause tachycardia so that hypotension will not be maximal. Myocardial ischaemia may occur.

Hypercapnia antagonizes the hypotension caused by ganglionic blocking drugs but potentiates that due to extra- or intradural sympathetic block.

The drug passes the placental barrier but does not cause ill effects in the foetus. Its action on the bronchi is not yet settled. Hypotension may stimulate pressor receptors in the walls of the aortic and carotid sinuses resulting in reflex bronchodilatation. On the other hand the release of sympathetic tone may favour bronchoconstriction and atelectasis has been described after its use. Hexamethonium and pentamethonium do not interfere with the anticurare effects of neostigmine.

Hypotension may cause alteration of cerebral function as shown by the flicker fusion test of Berg† while psychiatric disorders have been noticed after its prolonged use. A blood pressure below about 60 mm Hg has been seen to cause a turgidity and cyanosis of the liver by Bromage and death has been reported owing to liver necrosis.

**Renal Blood flow**—During general anaesthesia ganglionic blockade has no significant effect on renal blood flow because the glomerular filtration rate, the renal plasma flow and the flow of urine are already greatly diminished by the state of anaesthesia and are reduced in proportion to the depth of anaesthesia.

**Cerebral Blood flow**—Opinions vary about this, some investigators finding a reduced cerebral blood flow, others finding it well maintained due to decreased cerebrovascular resistance. In cerebral atheroma the vessels cannot relax to decrease vascular resistance and ischaemia may therefore occur in such patients. The brain is said to be more sensitive than the heart to acute hypotension‡.

**Coronary Blood flow**—The state of the coronary vessels probably plays a large part in the development of cardiac ischaemia when hypotension occurs. There is no evidence that hypotensive anaesthesia causes any permanent damage to the myocardium§ seen on electro-encephalograph evidence although marked T or ST wave alterations may indicate acute ischaemia.

P. van J. P. A. *Arch. int.* 1955 13 85.

† Nilsson, E. *Et J. Anæsth.* 1955 25 24.

‡ Robson, J. M. and Keele, C. A. *Recent Advances in Pharmacology* 1956 (2nd ed.) London: J. & A. Churchill.

§ Roll, S. W. N. and Cumming, A. R. R. *Anæsthesia* 1956 11 319.



### Ganglionic Blocking Agents *continued*

**Lung Changes**—Organized fibrinous pulmonary oedema with dyspnoea has followed the use of hexamethonium but only when it has been given over a long period

**Excretion**—The drug is not metabolized in the body but is excreted via the renal glomeruli 50 per cent of it in 2 hours and 90 per cent within twenty four hours after intravenous injection. If the blood pressure drops below about 50 mm Hg glomerular filtration ceases and excretion will no longer take place

### Pentolinium Tartrate (Pentapyrrolidinium Ansolysen M & B 2050A)

—This was synthesized in 1952 by Libman and used in anaesthesia by Enderby\*. Chemical composition is pentamethylene 1-5 bis (1 methyl pyrrolidinium). Its action is similar to that of hexamethonium but it is said to be five times more potent has an action slower in onset but longer in duration causes less tachycardia does not antagonize the neuromuscular blocking action of decamethonium and produces less parasympathetic ganglionic block than does hexamethonium (i.e. less tendency to ileus). Average intravenous dose is 10-20 mg in 0.5 per cent solution which takes about three minutes to produce a maximum fall in blood pressure

**Arfonad** (Trimetaphan camphorsulphonate)—This compound a thiophanium derivative was described by Randall and others in 1949 and is *d*-3,4-(1,3-dibenzyl-2-ketomidazolido)-1,2-trimethylene thiophanium *d*-camphorsulphonate (RO 1-222). It is supplied as the dry substance in 250 mg ampoules. It was used to reduce the blood pressure by Sarnoff in 1952 and reported on by Magill, Scurr and Wyman in 1953†. It is a ganglion blocking agent and has a direct dilator effect on peripheral vessels. It liberates histamine. During anaesthesia it can be given as an intravenous drip in a strength varying from 0.05 per cent to 0.2 per cent—0.1 per cent being the most commonly used (1 mg per ml). With this concentration the drip starts at 60 drops per minute (3-4 mg/min). The drug can be given as 5 per cent solution (the dose being 10 to 50 mg) by repeated single injections‡. It inhibits pseudocholinesterase. Hypotension depends on the speed of the drip and after the drip is stopped recovery is usually rapid. It is incompatible with thiopentone and other strongly alkaline solutions gallamine triethiodide iodides and bromides its hypotensive effect is antagonized by methylamphetamine and similar pressor drugs. It causes less tachycardia than does hexamethonium and causes more hypotension in arteriosclerotic patients than in those with normal blood pressure interferes with the heat regulating mechanism causing hypothermia after long operations and reduces the need for anaesthesia once a low pressure has been established. It may however cause tachyphylaxis tachycardia and rather prolonged hypotension.

Enderby G. E. H. *Lancet* 1954 2 1097

† Magill I. W., Scurr C. F., and Wyman A. J. B. *Ibid* 1953 1 217

‡ Kilduff C. J. *Ibid* 1954 1 337

It would seem that this drug with its rapid onset and short duration of action is a great improvement on the longer acting ganglionic blocking agents

Reserpine 1 to 2.5 mg intravenously half to two hours before operation potentiates the hypotensive action of trimetaphan

**Ecolid** (Su 3088 Chlorisondamine dimethochloride) — This powerful drug is at present under trial it was introduced by Plummer and by Grimson

**Azamethonium Bromide** (Isonidomid Pentamethyl diethyl 3 azapentane 1-5 diammonium dibromide) — This was first used by Ikin and Meier in 1950 and has some vogue on the Continent Its action is similar to that of hexamethonium Dose 10 mg each minute for five to ten doses

**Dibenzylene** (N-phenoxisopropyl-N-benzyl-β-chloroethylamine hydrochloride) — This is one of the β halo alkylamine group of adrenergic blocking agents allied to dibenamine and has been used\* to lower the blood pressure when hexamethonium has failed to do this It does not reverse the tachycardia It is dissolved in propylene glycol and alcohol and the dose is 12.5 to 100 mg

**Trophenium** (Phenactropinium chloride) — This new drug which is phenacyl homatropinium chloride was investigated by Johnston and Spencer in 1956 and described clinically as a hypotensive agent in anaesthesia by Robertson Gillics and Spencer (Robertson J D Gillics John and Spencer B I N *Brit J Anaesth* 1957 29 342)

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In spite of its chemical constitution trophenium does not inhibit structures innervated by post ganglionic cholinergic nerves i.e. it has no atropine like action

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Enderby C. F. H. *La. et al.* 1954 2 1027  
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Ganglionic Blocking Agents—Uses of Hypotensive Agents *continued*

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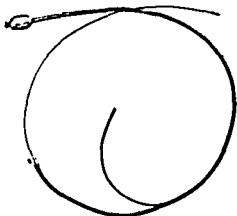
When a normal patient stands up after lying down his cardiac output is lessened and a reflex is initiated which causes vasoconstriction to maintain blood pressure. After general anaesthesia and still more after the use of hexamethonium this reflex vasoconstriction does not occur and so cardiac output being reduced because of the position profound hypotension results. The technique of controlled hypotension aims to block the sympathetic ganglia so causing hypotension. Posture is used to make the site of operation ischæmic. A systolic blood pressure between 60 and 80 mm Hg is aimed at. A steep upward tilt of the head is potentially dangerous as it may cause cerebral ischæmia and hypoxia while a steep head down tilt may cause venous congestion in the brain.

The technique is simple but must be meticulous. Neither the airway nor the patient's ventilation must be other than first class and afferent impulses must be properly blocked. Any of the ordinary anaesthetic combinations may be used such as thiopentone, pethidine, relaxant, trilete and minimal ether. It is often convenient to insert a Nit hell needle into a vein for intravenous injections. As full oxygenation is essential endotracheal intubation is usually employed. Before induction the blood pressure is taken and it is again taken after the anaesthesia has become stabilized. The patient is then placed in the optimal position for operation and a third blood pressure reading is made. A labile blood pressure is an indication that small dosage will probably be adequate. Other such indications for small dosage are increasing age and low metabolic rate. Arterio-sclerotics require a small dose and little posture to get a profound effect while young fit patients besides often developing tachycardia require a steep tilt and high initial dosage to show a good ischæmia. Posture is important in the attainment of good ischæmia the following being most helpful: supine with head elevated, prone, jack knife, lateral jack knife. In upper abdominal operations the elevation of the gall bladder bridge lowers blood pressure as also does positive pressure breathing and the actual opening of the peritoneal cavity. In lower abdominal operations ischæmia is not easy to produce nor is it when the patient is in the lithotomy position.

The blood pressure is taken at regular and frequent intervals. Facilities for blood transfusion must be to hand as bleeding is a dangerous complication because the normal response to it vasoconstriction is impossible. Great care and constant attention must be given throughout the operation and afterwards to the patient who is taken near to the point of death.

Suggested initial doses which should be given with the patient horizontal are \* (1) For normotensives 100-200 mg of

**A BRONCHIAL OCCLUSION WITH CUFFED SUCTION CATHETER AND ENDOTRACHEAL ANÆSTHESIA** with Thompson or Magill bronchus blocker (*Fig. 63*) placed in the bronchus of the affected lobe. The catheter and balloon must be accurately placed using a metal stylet and retained in position. The technique differs from that used for pneumonectomy in that the balloon is placed distal to the upper lobe bronchus. An upper lobe bronchus can be blocked with a Magill blocker but it is difficult and blockers are much more satisfactory in lower



*Fig. 63*—Magill bronchus occluder (Medical and Industrial Equipment Ltd.)

lobectomies. When inserting a cuffed suction catheter for a right lower lobectomy the middle lobe bronchus may be occluded so the balloon must be inserted deeply so that the middle and dorsal lobe bronchi will be free. For insertion of the bronchoscope in these cases the head of the patient must be on his opposite shoulder and usually a general anæsthetic is needed.

When there is much sputum in the bronchial tree it may be unwise to allow the lung to collapse during a thoracotomy as sputum in the bronchial orifice may prevent re-inflation of the lobe.

#### **B. ENDOTRACHEAL ANÆSTHESIA AND GRAVITY DRAINAGE**

**AGE**—After insertion of an endotracheal tube its proximal end is brought through a rubber obturator in the face mask (*Fig. 64*). The table is tilted to a steep angle of 35° for left thoracotomy (55° for right thoracotomy) so that secretions run into the trachea instead of into the dependent (sound) main bronchus if the patient is in the lateral position. If he is in the prone position (posterolateral incision) a 10° tilt is required. Secretions in the endotracheal tube can be sucked out when necessary; secretions passing outside the tube collect in the face mask. A balloon is not used as it would prevent this gravity drainage.

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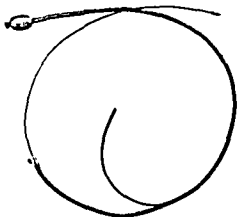
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under consideration is said to have two great advantages it reduces bleeding and prevents shock.

When a normal patient stands up after lying down his cardiac output is lessened and a reflex is initiated which causes vasoconstriction to maintain blood pressure. After general anaesthesia and still more after the use of hexamethonium this reflex vasoconstriction does not occur and so cardiac output being reduced because of the position profound hypotension results. The technique of controlled hypotension aims to block the sympathetic ganglia so causing hypotension. Posture is used to make the site of operation ischæmic. A systolic blood pressure between 60 and 80 mm Hg is aimed at. A steep upward tilt of the head is potentially dangerous as it may cause cerebral ischæmia and hypoxia while a steep head down tilt may cause venous congestion in the brain.

The technique is simple but must be meticulous. Neither the airway nor the patient's ventilation must be other than first class and afferent impulses must be properly blocked. Any of the ordinary anaesthetic combinations may be used such as thiopentone pethidine relaxant trilete and minimal ether. It is often convenient to insert a Mitchell needle into a vein for intravenous injections. As full oxygenation is essential endotracheal intubation is usually employed. Before induction the blood pressure is taken and it is again taken after the anaesthesia has become stabilized. The patient is then placed in the optimal position for operation and a third blood pressure reading is made. A labile blood pressure is an indication that small dosage will probably be adequate. Other such indications for small dosage are increasing age and low metabolic rate. Arteriosclerotics require a small dose and little posture to get a profound effect while young fit patients besides often developing tachycardia require a steep tilt and high initial dosage to show a good ischæmia. Posture is important in the attainment of good ischæmia the following being most helpful: supine with head elevated, prone, jack knife, lateral jack knife. In upper abdominal operations the elevation of the gall bladder bridge lowers blood pressure as also does positive pressure breathing and the actual opening of the peritoneal cavity. In lower abdominal operations ischæmia is not easy to produce nor is it when the patient is in the lithotomy position.

The blood pressure is taken at regular and frequent intervals. Facilities for blood transfusion must be to hand as bleeding is a dangerous complication because the normal response to it vasoconstriction is impossible. Great care and constant attention must be given throughout the operation and afterwards to the patient who is taken near to the point of death.

Suggested initial doses which should be given with the patient horizontal are \* (1) For normotensives 100-200 mg of

hexamethonium or 10-20 mg of pentolinium or 30-50 mg of arfonad ( ) 1 or hypertensives and patients over 55 years of age 50-80 mg of hexamethonium 5-10 mg of pentolinium or 20-30 mg of arfonad. The initial dose is the most important. Blood pressure can be further lowered by (a) A head up tilt (b) Abolition of the respiratory pump mechanism by controlled respiration thus reducing venous return and cardiac output (c) Slight positive pressure during controlled respiration augmenting this effect (d) Hypocapnia the result of hyperventilation. In the elderly each agent causes severe hypotension with little tachycardia. Other workers employ a dosage scheme such as the following—using hexamethonium (i) To young adults 50 mg (ii) To patients between 40 and 60 30 to 40 mg (iii) To hypertensives arteriosclerotics and those over 60 20 mg. Three minutes after injection the effects are assessed and more drug is given if required then or later usually in doses of 10 to 5 mg. The first dose is however the important one. A large initial dose causes maximal hypotension but may be dangerous in inexperienced hands. The last injection of hexamethonium should be given not less than twenty minutes before the end of the operation.

Failure to produce good ischaemia with ganglionic blocking agents may be due to (a) Incomplete block of sympathetic ganglia (b) Increased cardiac output due to tachycardia (c) Presence of pressor agents e.g. noradrenaline or adrenaline in the circulation (d) The presence of a phaeochromocytoma.

Signs of excessive hypotension include (1) A completely dry wound (2) Irregular respiration (3) The onset of cardiac irregularity.

To raise the blood pressure at the end of the operation or before that if necessary a return to the horizontal position is usually sufficient. Hypertensive drugs and also infusions may be used. Methedrine 4-8 mg intravenously is satisfactory but may cause cerebral stimulation and restlessness. Phenylephrine, methoxamine and noradrenaline are also used. Pressor drugs do not however restore vasomotor control. Towards the end of the operation the blood pressure is allowed to rise slowly and no patient should leave the table unless his blood pressure is 80 mm Hg. The journey back to the ward is made on a tilting trolley and if necessary the head-down position is employed for the first few hours. Blood pressure readings must be taken frequently in the post-operative period.

The results in successful cases are (1) A bloodless field of operation (2) Reduction of operative shock (3) Reduction of vomiting after operation (4) Delayed recovery from anaesthesia.

**The Dangers**—Hunter states that the technique should be confined to those cases where it makes the impossible possible. It should not be used to make the possible easy. The technique is at present experimental and has not found its true and permanent uses. Hypothermia decreases the dangers of hypotension.

*Anæsthesia for Lobectomy—Carlens's Double Lumen Catheter continued*

above this again is an inflatable tracheal cuff. Pilot bags are fitted to the two inflating tubes. Produced in three sizes 37, 39, 41 (French) each lumen approximating to Magill size 5, 6 and 7. It is said to have the following advantages: (1) It isolates the healthy lung from spread of disease. (2) It enables the anæsthetist to aspirate pus or blood from each lung without interrupting the anæsthesia. (3) Each lung can be inflated or deflated independently. During a left pneumonectomy closure of the left main bronchus is carried out (after aspiration) following the deflation of the balloons and withdrawal of the tube for 5 cm. The proximal cuff is re-inflated and the apparatus then used as a single lumen endotracheal tube. When there is stenosis or obstruction of the left main bronchus the tube cannot be used. It is passed blindly through a laryngoscope and adequate air entry into the lungs confirms the correctness of its position. The relatively small size of the lumina results in underventilation if respiration is spontaneous but this does not occur if a mechanical respirator is used. The tube is not unduly traumatic.

2. **THE COMBINED ENDOTRACHEAL TUBE AND BRONCHUS BLOCKER OF STUERTZBECHER\*** (Fig. 66).—An invention from Federal Germany, this consists of an endobronchial blocker with inflatable cuff projecting 7 cm. from the distal opening of a cuffed endotracheal tube. Each cuff has a pilot balloon while a third channel permits aspiration from the end of the blocker. This has a small metal tip for radiographic localization. A stilette of curved wire aids correct insertion through the laryngoscope and correct positioning is checked by auscultation and by X-ray localization. It enables (a) Either the whole lung or the part to be resected to be isolated from healthy lung tissue. (b) Secretions to be aspirated without interfering with ventilation. (c) The blocked off parts of the lung to be expanded or collapsed thus aiding identification.
3. **THE VELLACOTT ENDOBRONCHIAL TUBE†**.—This has been designed for use in cases of right upper lobectomy in patients with considerable amounts of sputum in whom it is impossible to place a Magill blocker in the right upper lobe bronchus. The tube of single lumen is stiffened by a wire. It has a proximal inflatable cuff in the trachea and a distal one opposite the right upper lobe bronchus which it occludes. Between these cuffs is a deeply cut gap in the wall of the tube which allows aeration of the left lung through its main bronchus. The tube is inserted over a bronchoscope.
4. **THE MACINTOSH AND LEATHERDALE TUBES‡** (Figs. 67, 68).—Two tubes have been designed for insertion into the bronchi through a laryngoscope. (a) A left endobronchial tube with inflatable cuffs designed to lie in the left main bronchus

Och. S. R. An. J. 157 955 16 474  
 † Vellacott W. H. B. J. A. N. 1951 26 442  
 ‡ Macintosh R. R. and Leatherdale R. A. L. J. 1955 27 556

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Oech S R *Anæsthesia* p. 955 18 463

† Vellacott, W H B & J *Anæsthesia* 1954 26 442

‡ Macintosh, R R & Leatherdale J R A L *Ibid.* 1955 27 536

## CHAPTER VIII

## EXPLOSION RISKS IN ANÆSTHESIA\*

Under average conditions explosive anæsthetic mixtures become diluted with the air of the theatre to a non-flammable range before reaching a distance of 2 ft. from a point of leak of anæsthetic vapours involving the quantities of these vapours used in ordinary surgical procedures.

The following table published by the U.S. Bureau of Mines shows the limits of flammability of anæsthetics —

	<i>In Air</i> Per cent	<i>In Oxygen</i> Per cent	<i>In Nitrous Oxide</i> Per cent	<i>Density</i> Air = 1
Ethylene	3.05 to 28.6	2.9 to 29.9	1.9 to 40.7	0.97
Cyclopropane	2.4 to 10.5	2.45 to 11.0	1.4 to 30.5	1.45
Ethyl chloride	4.0 to 14.8	4.05 to 15.2	2.1 to 22.5	2.23
Diethyl ether	1.7 to 27.0	1.85 to 85.5	1.4 to 24.8	2.42
Methyl ether	1.85 to 13.5	2.1 to 82.0	1.5 to 24.2	2.56

Before an explosion can occur a source of heat necessary to raise a liquid to its flash point or a vapour to its ignition temperature is required.

**Ether**—Mixtures of gas oxygen and ether are always explosive and more dangerous than ether and oxygen alone. Pure ether vapour will not explode. The addition of reasonable percentages of carbon dioxide will not prevent explosion. Ether vapour is made more dangerous when mixed with oxygen or nitrous oxide than when mixed with air. Ether in air burns slowly the flame being unlikely to travel to the patient's air passages. Ether vapour is heavier than air and so sinks to the floor. A source of ignition may be safe if it is held above the ether-air mixture as on an open mask providing no draughts spread the ether vapour. The presence of peroxides will enable it to ignite at still lower temperatures. Ether peroxides are less volatile than ether and so may accumulate in ether bottles or anæsthetic machines if residual ether is not frequently discarded. Ether will not ignite spontaneously. Hot wires and surfaces—below the temperature of visible dull red heat (i.e. 300 C)—may set going an invisible cool flame in ether vapour. This may travel.

If the administration of ether is discontinued five minutes before a possible source of ignition is exhibited the patient's exhalations are unlikely to burn or explode. Further anæsthesia can be maintained with a non-explosive agent. Open ether (without added oxygen) has never been known to cause injury to a patient from ignition.

*See Report of a Working Party on Anæsthetic Explosions including Safety Code for Equipping and Installation (Anæsthetic machines: Drs. Marston, Low, Morton and Gallely) London: H.M.S.O. 1956. 6d. Also Physics for the Anæsthetist 2nd ed. 1958 Macintosh, M. M. Mushin, W. W. and Copst. H. G. Oxford: Blackwell.*

**Ganglionic Blocking Agents Dangers of continued**

Trouble has been reported from cerebral and coronary thrombosis renal and hepatic ischæmia reactionary hæmorrhage ileus cerebral damage arterial thrombosis (e.g. the carotid and limb arteries) and massive atelectasis. Primary heart failure is an ever present anxiety.

In the cat hexamethonium and also trimetaphan can cause neuro muscular block if the animal has been previously given mecamylamine so this state of affairs might arise in patients under treatment for hypertension.\*

**Indications**—These are not yet established but may include the following—

- 1 Neurosurgery especially in operations for vascular tumours and aneurysms. Hypotension causes shrinkage of the brain.
- 2 Peripheral vascular surgery e.g. coarctation of the aorta.
- 3 Removal of vascular tumours.
- 4 Operations associated with voluminous hæmorrhage.
- 5 Plastic surgery.
- 6 Fenestration operations tympanoplasty etc.
- 7 When a patient's abnormal blood group makes transfusion difficult.
- 8 The possible prevention of shock by autonomic paralysis.

**Contra indications**—When anæsthetic technique or skill is not of a high order.

In patients with coronary disease (this is not always detectable before operation). Shock poor renal or hepatic function cerebral vascular abnormality conditions causing hypotonia Addison's disease in pregnant women.

### **POTENTIATION OF HEXAMETHONIUM BY PROCAINE AMIDE**

Mason and Peimoret† have reported on their use of procaine amide in association with hexamethonium in those patients who develop a tachycardia with indifferent hypotension after hexamethonium alone. Procaine and its amide antagonize the stimulating action of acetylcholine on the ganglion cells and at the same time decrease the amount of acetylcholine liberated by the postganglionic endings so the ganglionic inhibition is further increased. Procaine amide does not achieve its effect by a direct cardiac action. Intravenous injection of 0.5–1 g. is given when hexamethonium causes a tachycardia greater than 110 beats per minute. By this means ischæmia is greatly increased and complications have so far not been noticed.

There are workers‡ who claim better results for certain types of operation e.g. fenestrations by the use of phenothiazine derivatives than by ganglion blocking agents i.e. there is less bleeding.

The pros and cons of induced hypotension are well set out in the *Brit med Bull* 1958 14 1 49 53 by Enderby G E H (pro) and Armstrong Davison M H (con).

\* P yn J P *Br J Anesth* 1957 29 358.  
 † Mason, A. A. & Peimoret J P *Br Med J* 1955 1 250.  
 ‡ Mason, S. A. *Pac World Congr J Anesthesiologist* 1956 243. Minneapolis Du gross Publishing Co.

The way to banish the danger from static electricity is to arrange for static charges to be carried away. Each and every person standing on a floor should be the common link with the earth which is a fact, etc. in a theatre are interjected. A person in the street would have an electrical resistance between his feet and ground of 25 million ohms when measured between two feet being 2 ft 3 in apart at any point on the floor. If a conductivity having been attained it becomes necessary for all equipment and personnel to be brought into electrical continuity with it.

One obvious way of preventing the development of static electricity would be to eliminate non-conductors and wherever possible substitutes for easily electrified materials should be used. The following items are all charge producers and non-conductors: rubber mattresses, plastic sheet material, plastic caps, plastic pillow covers, woollen blankets, cotton sheets, woollen suits, rayon and nylon garments and hosiery, cotton overalls and gowns, rubber stool tops, painted stool tops, rubber gloves, anæsthetic reservoir bags. The next group comprises commonly used insulators: non-conductive castors on tables and trolleys, non-conductive rubber stool tips, interior non-conducting parts of anæsthetic machines. The following are both charge producers and insulators: non-conductive floors, non-conductive shoes, non-conductive rubber tyres, non-conductive rubber tubes of anæsthetic machines and anæsthetic masks. Undergarments are not included in the list as they do not add to a person's electrostatic charge when covered with a cotton gown.

To ensure the neutralization of all charges—with consequent avoidance of sparks—the non-conductive material should be bridged by suitable conductors to a conducting floor. One easy way of lessening danger in the absence of conducting rubber is to connect the metal parts of the table anæsthetic machine and anæsthetist's stool with the floor by means of a wet towel which itself makes contact with the anæsthetist's leather soled shoes. This forms a makeshift electrical intercoupler. It is virtually impossible to design conditions in which a dangerous static discharge will never occur.

The Vapotester\* is essentially a Wheatstone bridge in which the electric current is balanced. It enables the anæsthetist to measure accurately the concentration of the anæsthetic agent and the explosibility of the mixture.

The Statometer\* detects the presence of static electricity, its source and intensity.

**SPONTANEOUS IGNITION**—This may occur if oil or grease is allowed to react with nitrous oxide or oxygen under pressure, e.g. when escaping from a cylinder. This absolutely contra-indicates the use of lubricants on reducing valves and gas cylinders.

Explosions can arise from ignition of such gases as hydrogen, methane and sulphuretted hydrogen arising from the stomach or bowel from fermentation†.



**Ethyl Chloride** —This burns actively if ignited and will then explode if its vapour is in an oxygen mixture

**Divinyl Ether** —Behaves in a similar manner to diethyl ether

**Cyclopropane** —This is very explosive when mixed with oxygen and can be ignited by a spark of less energy than that required to explode an ether-oxygen mixture. As cyclopropane is present in the tissues after 20–30 minutes diathermy should not be used in body cavities when this anæsthetic is being used. Nor must diathermy be applied to the lungs. The possibility of broncho-pleural fistula must be borne in mind.

**Trichlorethylene** —This is not flammable in air under operating theatre conditions. A vapour strength of or over 10 per cent in air enriched with oxygen is flammable but such a strength is not likely to be used when it is considered that a vaporizing bottle of a Boyle machine with the plunger up is unlikely to deliver a concentration greater than 1.5 per cent.

**Halothane** —Is non flammable and non-explosive

**Trifluoroethyl Vinyl Ether** —Lower flammability limit in air oxygen or nitrous oxide and oxygen is 4 per cent

**Oxygen** —If oxygen under pressure comes into contact with oil or grease an explosion may occur. Thus no oil must be used on valves etc. The same applies to nitrous oxide and oil or grease.

### Sources of Ignition —

- 1 **HEAT** —From open flames or fires from hot surfaces or wires from overheating electric bulbs e.g. on endoscopes from thermocautery. Pipes lighters cigarettes. The minimum temperature which will ignite an explosive anæsthetic mixture is stated to be 180° C (355° F).

- 2 **ELECTRIC CURRENT** —(a) Normal (b) Faulty

a **NORMAL** —Diathermy which comes next in order of importance after static electricity as a cause of explosions. electric cautery sparks from motors sparks from X ray machines sparks from switches etc.

b **FAULTY** —Short circuits in electrical apparatus faulty wires and cables breaking of bulbs.

All portable electrical apparatus should be fitted with explosion proof switches and three wire flex the third wire being connected to the outer casing of the apparatus and to the third point in a wall plug. The plug should incorporate a locking device making it impossible to remove the plug while the current is switched on.

- 3 **STATIC ELECTRICITY** —The most frequent cause of explosions in operating theatres. To reduce the hazard of such explosions to a minimum major changes are required in some hospitals. Static electricity is produced when two dissimilar surfaces are brought into intimate contact and then separated. It also occurs just before the point of contact of a highly charged non-conductor with a conductor.

theatre covered by woollen blankets they should never be removed quickly and should be taken out of the theatre suite before the patient is anaesthetized. Woollen stockings should not be rapidly pulled off.

- 9 The relative humidity of the atmosphere should not be allowed to fall below 50 per cent. It may be most difficult to raise the humidity. Static sparks are more frequent when the air is dry and the barometric pressure high. Humidity of the atmosphere will not prevent explosions taking place in the anaesthetic apparatus.
- 10 Ignitable gas flow should be turned off if metallic unions have to be made or unmade during anaesthesia.
- 11 Avoid spilling ether etc. about the theatre.
- 12 Avoid cigarettes matches etc. in the anaesthetic room.
- 13 Remember that spirit lotions may ignite.
- 14 When diathermy is applied to the bladder hydrogen is given off and may ignite inside the bladder. Prolonged use of diathermy may cause a strong positive charge on the patient which may lead to spark formation unless he is adequately earthed. If the diathermy electrode is actually touching the tissues or the hamostat before the current is switched on arcing is minimized.
- 15 When there is risk of explosion use an intravenous barbiturate trilete gas-oxygen regional halothane or chloroform anaesthesia.
- 16 See that fire fighting equipment is available and in good order.
- 17 The use of a tube containing radon to neutralize static electricity\*. The use of a hair-dryer containing a radioactive salt has been recommended.
- 18 When cautery or diathermy is applied to the mouth or bowel nitrogen or carbon dioxide should be run in to wash out ignitable gases e.g. hydrogen and methane because they do not support combustion. It is possible that if all theatre suites were equipped with antistatic rubber exclusively and if all woollen blankets were prohibited explosions from static electricity the commonest cause of anaesthetic explosions would almost disappear.

This vast question is excellently discussed in a brochure published by the U.S. Bureau of Mines (Report of Investigations 4833) entitled *Static Electricity in Hospital Operating Suites Direct and Related Hazards and Pertinent Remedies* by P. G. Guest, V. W. Sikora and B. Lewis 1952. Pittsburgh Pa. U.S.A.

**Recommendations for Prevention \*—**

- 1 Explosive anæsthetics should not be used when the following potentially dangerous pieces of apparatus are employed diathermy near face cautery X rays electric motors electric and gas heating equipment—unless suitable precautions are taken. It is probably safe to give open ether in an anæsthetic room and then to bring in the anæsthetized patient for examination.
- 2 Administration of ignitable anæsthetics should be stopped 3–5 minutes before diathermy or cautery is used. Ether bottles should be removed—not merely turned off—and air should be blown through apparatus. Maintenance of anæsthesia can be by a non flammable agent.
- 3 Oxygen and nitrous oxide although not flammable increase the danger of other agents. Reducing valves should not be changed from one type of gas to another.
- 4 Small electric bulb should not be overrun. voltage should not be greater than the 2.5 to 6 volts for which they are designed. The use of batteries instead of electric mains prevents accidental overrunning.
- 5 Electric wiring and apparatus should be inspected frequently by an electrician even though their function is not impaired. All electrical apparatus should have a third wire for positive grounding.
- 6 Foot switches must be flame proof as ether vapour falls towards the floor. Wall plugs should be 5 ft above the floor if they are not flame proof. Electric connexions ideally should all be flame proof.
- 7 In all rooms where explosive anæsthetics may be used a conducting floor should be provided. This includes X ray rooms where the risk of electric shock from the high potentials used is remote. Swabbing theatre floor with 4 per cent calcium chloride solution is said to increase its electrical conductivity while damp sheets have the same effect. Wax polish must not be allowed on conducting floors.
- 8 Conducting rubber or vinyl thermoplastic products having antistatic properties should be used for tyres of trolleys tables stool tips etc. The resistance of antistatic material should be between 10 megohms and 50 000 ohms. For breathing tubes face masks reservoir bags. Where antistatic rubber is not available the risk can be reduced if the rubber is wetted internally and externally before use. For mattresses rubber sheeting aprons etc. Non-conducting mattresses can be made reasonably safe if they are completely enclosed in a close fitting cover of such antistatic material as cotton linen or viscose rayon. For rubber floors. All personnel should wear either conducting rubber or leather footwear each shoe having a resistance between 0.1 and 1 megohms. cotton blankets and sheets should replace woollen articles in the theatre suite and should not be allowed to become too dry. When a patient comes to the

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- 8 Conducting rubber or vinyl thermoplastic products having antistatic properties should be used for tyres of trolleys tables stool tips etc. The resistance of antistatic material should be between 10 megohms and 50 000 ohms. For breathing tubes face masks reservoir bags. Where antistatic rubber is not available the risk can be reduced if the rubber is wetted internally and externally before use. For mattresses rubber sheeting aprons etc. Non-conducting mattresses can be made reasonably safe if they are completely enclosed in a close fitting cover of such antistatic material as cotton linen or viscose rayon. For rubber floors. All personnel should wear either conducting rubber or leather footwear each shoe having a resistance between 0.1 and 1 megohms. cotton blankets and sheets should replace woollen articles in the theatre suite and should not be allowed to become too dry. When a patient comes to the

theatre covered by woollen blankets they should never be removed quickly and should be taken out of the theatre suite before the patient is anesthetized. Woollen stockings should not be rapidly pulled off.

- 9 The relative humidity of the atmosphere should not be allowed to fall below 50 per cent. It may be most difficult to raise the humidity. Static sparks are more frequent when the air is dry and the barometric pressure high. Humidity of the atmosphere will not prevent explosions taking place in the anesthetic apparatus.
- 10 Ignitable gas flow should be turned off if metallic unions have to be made or unmade during anesthesia.
- 11 Avoid spilling ether etc. about the theatre.
- 12 Avoid cigarettes matches etc. in the anesthetic room.
- 13 Remember that spirit lotions may ignite.
- 14 When diathermy is applied to the bladder hydrogen is given off and may ignite inside the bladder. Prolonged use of diathermy may cause a strong positive charge on the patient which may lead to spark formation unless he is adequately earthed. If the diathermy electrode is actually touching the tissues or the haemostat before the current is switched on arcing is minimized.
- 15 When there is risk of explosion use an intravenous barbiturate, trileal gas-oxygen, regional halothane or chloroform anesthesia.
- 16 See that fire fighting equipment is available and in good order.
- 17 The use of a tube containing radon to neutralize static electricity. The use of a hair-dryer containing a radioactive salt has been recommended.
- 18 When cautery or diathermy is applied to the mouth or bowel nitrogen or carbon dioxide should be run in to wash out ignitable gases e.g. hydrogen and methane because they do not support combustion. It is possible that if all theatre suites were equipped with antistatic rubber exclusively and if all woollen blankets were prohibited explosions from static electricity the commonest cause of anesthetic explosions would almost disappear.

This vast question is excellently discussed in a brochure published by the U.S. Bureau of Mines (Report of Investigations 4833) entitled *Static Electricity in Hospital Operating Suites, Direct and Related Hazards and Pertinent Remedies* by P. G. Guest, V. W. Sikora and B. Lewis 1952, Pittsburgh Pa. U.S.A.

**Recommendations for Prevention \*—**

- 1 Explosive anæsthetics should not be used when the following potentially dangerous pieces of apparatus are employed diathermy near face cautery X rays electric motors electric and gas heating equipment—unless suitable precautions are taken It is probably safe to give open ether in an anæsthetic room and then to bring in the anæsthetized patient for examination
- 2 Administration of ignitable anæsthetics should be stopped 3–5 minutes before diathermy or cautery is used Ether bottles should be removed—not merely turned off—and air should be blown through apparatus Maintenance of anæsthesia can be by a non flammable agent
- 3 Oxygen and nitrous oxide although not flammable increase the danger of other agents Reducing valves should not be changed from one type of gas to another
- 4 Small electric bulbs should not be overrun voltage should not be greater than the 2.5 to 6 volts for which they are designed The use of batteries instead of electric mains prevents accidental overrunning
- 5 Electric wiring and apparatus should be inspected frequently by an electrician even though their function is not impaired All electrical apparatus should have a third wire for positive grounding
- 6 Foot switches must be flame proof as ether vapour falls towards the floor Wall plugs should be 5 ft. above the floor if they are not flame proof Electric connexions ideally should all be flame proof
- 7 In all rooms where explosive anæsthetics may be used a conducting floor should be provided This includes X ray rooms where the risk of electric shock from the high potentials used is remote Swabbing theatre floor with 4 per cent calcium chloride solution is said to increase its electrical conductivity while damp sheets have the same effect Wax polish must not be allowed on conducting floors
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useful in children under 4. Shivering and vasoconstriction must be prevented by relaxants chlorpromazine or anaesthetics

## 2. Internal or Body Cavity Cooling —

- a Pleural when chilled saline is poured into the pleural cavity after thoracotomy
- b Gastric when a balloon is inserted into the stomach and ice-cold water is made to circulate through it\* in the anaesthetized patient. By this means the blood in the large abdominal vessels and in the neighbouring viscera is cooled

## 3 Extra corporeal Blood stream, or Pervascular Cooling —

This was originally arterio → venous but is now usually veno → venous employing the two venae cavae †‡ For cardiac surgery it has the advantages that it is not carried out until the heart has been examined under vision and the operative procedure definitely decided upon and must be confined to use in cardiac surgery. A catheter is inserted through the right auricular appendage into the superior vena cava blood is withdrawn from it passed by a rotary pump through a plastic cooling coil immersed in a jar containing a mixture of alcohol saline and solid carbon dioxide at a temperature of  $-2^{\circ}\text{C}$  § The blood after cooling is returned to the inferior vena cava via a catheter in the same incision in the right auricular appendage. Clotting does not occur in the tubing at low temperatures. Circulatory arrest can fairly safely last 5–10 minutes at  $28^{\circ}\text{C}$  and ventricular fibrillation is not common above this temperature. Below  $25^{\circ}\text{C}$  the ventricles tend to fibrillate spontaneously. The dangers of hypoxic brain damage are greater than those of ventricular fibrillation.

During hypothermia the temperature may be taken from the upper oesophagus (cardiac temperature) the pharynx (cerebral temperature) or the rectum. It should be above  $30^{\circ}\text{C}$  for neurosurgery (five to ten minutes of cerebral ischaemia) and around  $28^{\circ}\text{C}$  or a little lower if cardiac standstill for five minutes is planned.

During extra-corporeal cooling shivering is not seen while rewarming is simple. Japanese workers have recently taken blood from the carotid artery cooled it and returned it to the same vessel so cooling the brain more than other parts of the body. This allows a longer period of circulatory arrest during cardiac surgery with a decreased risk of ventricular fibrillation.

**Rewarming** — After surface cooling the patient can be allowed to regain heat normally or this can be expedited by warm blankets warm water warm air or the high frequency current. After internal cooling warm water or saline is used in the pleura or the intragastric balloon. After pervascular cooling the cooling coil can be immersed in saline or melted sodium thiosulphate crystals. It is usual to rewarm to  $35^{\circ}\text{C}$  or until consciousness has returned before sending the patient back to bed.

Khalil H H *Lancet* 1955 1 185  
 † Ross D N *Ibid* 1954 1 108 and *Br J Med* B II 1955 11 226  
 ‡ Brock, R. C. *Proc R Soc Med* 1956 49 347  
 § Lucas B G B *Ibid* 1956 49 345



## CHAPTER XXVII

## INDUCED HYPOTHERMIA

Induced hypothermia is a method used to lower the metabolism of the body as a whole and to reduce the dangers from hypoxia and the cellular damage resulting from regional occlusion of the circulation of the brain the heart the liver the kidneys and the legs. Cooling enables certain specialized tissues of the body to withstand periods of hypoxia which in its absence would cause harm. It has also been used to treat hyperpyrexia during and after operation and to treat shock.

**History**—1905 Simpson and Herring coined the term artificial hibernation and showed that cold—below about 28 C—could act as a general anæsthetic.

1938 Temple Fay treated carcinoma by lowering the body temperature—cryotherapy.

1947 Delorme of Edinburgh showed that hæmorrhagic shock was better tolerated by cooled dogs than by dogs at normal temperatures.

1950 Bigelow and his colleagues from Toronto\* showed that with progressive cooling the rectal temperature and the oxygen consumption of the body showed an almost linear relationship that even slight shivering doubled oxygen consumption and that no oxygen debt was incurred by the tissues. Surface cooling was the method used.

1951 Blood stream or perivascular cooling employed by Delorme of Edinburgh and by Boerema of Holland.

1953 Pioneer work was done by Swan, Virtue and their colleagues in Denver.

The so-called artificial hibernation of Laborit and Huguenard in France (1951) using chlorpromazine etc. should not be confused with induced hypothermia.

## TECHNIQUES OF HYPOTHERMIA

- 1 **Surface Cooling**—The original method of Bigelow and the simplest method. The patient is anæsthetized intubated and then placed in a cold bath on a refrigerated mattress wrapped in a refrigerated blanket sprayed with cold water and exposed to a fan draught or surrounded by ice bags or encased in a box into which cold air is blown. Time taken to reduce the temperature to 30–26 C is between three-quarters of an hour and three hours—largely depending on the size of the patient. When removed from the cool environment the patient's temperature may drop a few degrees further and this is worse in obese and large patients—the so-called after-cooling which may be dangerous by causing ventricular fibrillation in certain cases. The cold bath method is

- (4) Defective coronary circulation\* Treatment involves
- (1) Restoration of cerebral circulation by cardiac compression
  - (2) Restoration of heart beat If this occurs during an operation on the heart and if the temperature is low enough to abolish circulation temporarily the cardiectomy is performed and defibrillation left until the operative intervention is complete In other cases cooling is terminated the heart massaged to make it tonic and then it is electrically depolarized or defibrillated A noradrenaline drip may be required to sustain the blood pressure Intracoronary injection of neostigmine vigorous hyperventilation and avoidance of temperature below 28° C reduce the likelihood of this complication
- 2 TENDENCY TO HÆMORRHAGE—Uncontrolled oozing is sometimes seen after hypothermia especially if it has been prolonged It is due to a reduced coagulability of the blood perhaps due to a high heparin level It is also present when an artificial pump oxygenator is used Damage to platelets may also be a factor in causation
- 3 BRAIN DAMAGE—This may follow a too prolonged interruption of the cerebral circulation
- 4 ATLECTASIS—May occur
- 5 HISTOPATHOLOGICAL CHANGES—These have been reported to occur in vital organs †

**Uses of Hypothermia**—As the technique is still partly experimental indications are not as yet agreed on It has been used—

- 1 In neurosurgery to allow the brain to be deprived of blood in whole or in part for 5–10 minutes Or to make hypotension safer
- 2 In cardiac surgery repair of atrial septal defects and of pulmonary and aortic valvular stenosis to prevent hypoxic damage and to reduce hæmorrhage In the future the artificial pump oxygenator or cardiopulmonary by pass may displace it in cardiac surgery ‡
- 3 To enable the circulation to be interrupted during operations on the great vessels the liver kidneys or legs for periods over ninety minutes For operations on vessels distal to the origins of the renal arteries hypothermia is unnecessary
- 4 To reduce the dangers of hypotension during certain otorhino logical plastic and neurosurgical operations and those operations requiring thoracotomy in the presence of coarctation of the aorta §
- 5 To reduce the ill effects of shock
- 6 To retard harmful reaction to brain injury|| and to prevent and treat hyperpyrexia occurring in such cases During operations on the brain stem even when not associated with arterial occlusion or hypotension ¶

\*Lucas B G B *Brit med Bull* 1958 14 1 47

†Knock P *Lancet* 1955 2 837

‡Cleland W P and others *Brit med J* 1958 2 1369

§Gr J T C *Lancet* 1957 1 383

||Rowbotham G F and others *Ibid* 1957 1 1016

¶Inghis J M and Turner E *Ibid* 1957 1 1335

**Techniques of Hypothermia continued****Effects of Hypothermia —**

- 1 **THE CARDIOVASCULAR SYSTEM**—Arrhythmias occur at temperatures below 30 C spontaneous ventricular fibrillation may be seen but is not likely above 28 C Bradycardia not due to vagal overaction is progressive The blood pressure and pulse rate fall progressively as the temperature gets lower An intravenous drip of trimetaphan camphosulphonate (arfonad) is said to abolish arrhythmias \*
- 2 **RESPIRATION**—At 32 C the oxygen requirement is reduced by 30 per cent and at 30 C by 50 per cent The oxygen dissociation curve is shifted to the left so that liberation of oxygen to the tissues is hindered The respiration rate falls and breathing ceases at a temperature of 26 C An acidosis results but is probably metabolic and not respiratory in origin Respiratory acidosis should be prevented by hyperventilation and this should be continuous and not spasmodic with sudden changes During neurosurgical operations however normal respiration may be allowed † and respiratory acidosis need not occur
- 3 **CENTRAL NERVOUS SYSTEM**—The cerebral cortex can tolerate the acute hypoxia due to complete circulatory arrest for 5–10 minutes at a temperature of 28 C There is a reduction in brain volume and cerebrospinal fluid pressure changes appreciated by the neurosurgeon
- 4 **HEPATIC AND RENAL FUNCTION**—The function of these organs is depressed during hypothermia so that intravenous agents e.g. glucose thiopentone gallamine etc. which are excreted via the kidneys must be given in small amounts Cellular damage in the liver kidneys and suprarenals has been reported

**Methods of Anaesthesia during Hypothermia**—During the period of cooling and rewarming a careful electro encephalograph record is taken and the temperature is constantly observed from thermometers and thermocouples placed in the oesophagus the pharynx the rectum and on the skin of the forehead Some workers employ chlorpromazine as it prevents shivering causes vasodilatation and aids dissipation of heat reduces muscle tone and depresses the heat regulating centre Various anaesthetic techniques have been used including the thiopentone pethidine gas oxygen relaxant sequence ether and cyclopropane Only light anaesthesia is required and this together with a relaxant abolishes shivering Unconsciousness is not always caused by hypothermia to 28 C Hyperventilation reduces arrhythmias sometimes associated with respiratory acidosis

**Complications of Hypothermia —**

- 1 **VENTRICULAR FIBRILLATION**—Causes (1) Deep anaesthesia (2) Respiratory acidosis (3) Physical irritation of heart

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Inhalations of carbon dioxide have no place in the treatment of post-operative respiratory depression but can be given for short periods to cause hyperpnea and so expedite the excretion of volatile anaesthetics

An excellent position recommended by Macintosh for adoption after all operations is that usually adopted solely for patients returning to bed after operations on the upper respiratory tract i.e. semi prone with lower arm posteriorly and a pillow anteriorly upper leg flexed at knee joint

**2 Position of Arms and Legs**—Pressure on and stretching of nerves of arm must be avoided by care of the arms. The following methods may be used during operation

- a Each arm can be well tucked under the buttock palm down
- b A draw sheet is passed under the back and over the arms which are at the side of the body. The free ends are then firmly tucked under the buttocks imprisoning the arms
- c Wrist straps are firmly attached to a broad strap surrounding the table or to the table direct
- d Arms can be securely wrapped up at the side of the chest with elbows flexed in the patient's nightgown. This is useful when the gall bladder bridge is to be used
- e One or both arms are abducted to a right angle anterior to the coronal plane of the body and secured to a padded arm table or padded board. Arm rests unattached to the table are undesirable as they do not move with the table. This method is most useful if frequent estimations of blood pressure are to be made or if intravenous medication is likely to be necessary during the operation

Firm fixation is especially necessary if light anaesthesia is to be employed or if the patient is to be operated on in the conscious state. In these cases in addition a padded strap should be passed just above the knees and firmly secured beneath the table

When the lithotomy position is required both legs should be moved together to avoid strain on the pelvic ligaments

Legs should lie flat on the table not crossed one over the other. The tendo Achillis must not rest on the unpadded edge of the table. A soft pad raising heels from the table avoids pressure on the calf veins and so may lessen the incidence of thrombosis occurring at this site

**3 Moving the Patient**—Anaesthetized patients stand moving badly this is especially so when the blood pressure is low. All movements should be smooth and gentle not jerky and rough. If a canvas stretcher is not available three people should if possible lift the patient who should be rotated on to his side face to the lifters as soon as he is lifted from the bed or trolley

For operations in the lithotomy position the patient should be placed supine on the table with his feet on an adjustable stool and his anterior superior iliac spines on a level with the break in the table. Then when the knees are flexed the position will be correct without further tugging down of the patient

*Uses of Hypothermia continued*

- 7 In children to prevent the serious complications of hyperpyrexia and tachycardia associated with anaesthesia general disease e.g. poliomyelitis
- 8 To reduce the hyperpyrexia sometimes seen as the result of hypoxic cerebral damage in patients who have sustained cardiac stand still on the operating table
- 9 In thyrotoxic crises

Extracorporeal cooling is almost confined to cardiological work surface cooling is usually preferred for other conditions when hypothermia is indicated

Deliberate asystole has been induced by the intracoronary injection of 1 ml of 5 per cent potassium chloride per mg of body weight or by the intra aortic injection of 10 mg/kg of acetylcholine

Hypothermia presents many problems and views as to its utility techniques and indications have not been crystallized It should never be undertaken lightly \*

## CHAPTER XXVIII

## MANAGEMENT OF THE UNCONSCIOUS PATIENT†

- 1 **Airway**—All mucus and blood etc must be aspirated from the air passages before the patient leaves the table Similarly solid foreign matter e.g. vomitus should be removed via a bronchoscope without delay if its presence is suspected

If maintenance of the airway is difficult an oropharyngeal tube should be inserted before the patient leaves the table An endotracheal tube can be removed either in the theatre and replaced by a pharyngeal airway or later by the ward sister when the cough reflex has returned In patients likely to be comatose for long periods a tracheostomy is preferable to an endotracheal tube which should probably be removed after 36 hours

The anaesthetist must satisfy himself that the attendants taking the patient from the operating table back to his bed are capable of maintaining a good airway by manipulation of the lower jaw or failing this by the use of a gag and tongue traction It is of course very important that the patient should not leave the anaesthetist's care until he is able to ventilate himself properly

See also the following articles: Scott C. P. *Proc R Soc Med* 1955 48 107 L. Cus, B. G. B. *Ibid.* 1956 49 345 Ros. D. N. *Ibid.* 1956 49 365 Brock. H. *Ibid.* 1956 49 347 Gray T. C. *Lancet* 1957 1 354 Gray T. C. *Med J* 1957 Special Article 175 A. notat on *Lancet* 1957 1 675 and Malinow I. W. Gray T. C. and Davies, S. *Ibid.* 1958 2 1196.

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*Moving the Patient continued*

For patients who have been in the Trendelenburg position during operation and in patients suffering from circulatory depression a trolley which can maintain a head down tilt should be used to take the patient back to bed

- 4 Position in Bed** —The patient should lie in the semi prone position until his reflexes return This is maintained by a pillow between the bed and the chest the lower arm is placed behind the trunk the upper knee is flexed This helps to maintain a free airway by causing the tongue to fall away from the posterior pharyngeal wall it also helps to prevent aspiration of vomitus into the air passages

About 20 per cent of the deaths associated with anaesthesia occur during the first thirty minutes after operation

## CHAPTER XXIX

## ANÆSTHETIC RECORDS

Record cards should be used in all major operations They are doubly useful they enable the anaesthetist to assess accurately the condition of the patient during the operation they are invaluable for reference

Nosworthy's cards are most useful and convenient and three types are now published by the Copeland Chatterson Co They enable the sex age physical state pre operative complications anaesthetic techniques complications during anaesthesia plane of anaesthesia site of operation duration of operation premedication anaesthetics used post-operative complications etc to be accurately charted while they allow for a 5 minute record of the pulse and blood pressure readings By converting a series of circles into notches rapid sorting is possible with the aid of a knitting needle

Pulse readings should be made from a watch with large second hand or better still from a stop watch

The rubber tubes of the sphygmomanometer must be ample in length while the receiving end of the phonendoscope must be carefully and accurately applied to the arm over the brachial artery If application is made 2 in above the elbow joint the cuff of the sphygmomanometer can be applied over the phonendoscope helping to prevent the latter from slipping

In every case the agents doses and methods of administration should be written in the operation book followed by the anaesthetist's signature

## CHAPTER XXX

## THE ANÆSTHETIC OUT-PATIENT CLINIC

This should be attached to each hospital group to prevent the anaesthetist being faced by a patient in the ward admitted from the waiting list who is not in the optimal state for surgery. For the anaesthetist to see the patient a day or two before operation is not enough—he should be interviewed as soon as his name is placed on the waiting list if his operation is likely to be of a major nature or if his health is not beyond reproach.

The following should receive attention —

1. **THE RESPIRATORY SYSTEM**—No patient should undergo a non urgent operation while he is suffering from an acute infection of the upper respiratory tract. Chronic chest disease is more difficult to control but much can be done.

*Smoking should be completely given up for the three weeks before operation.* There is statistical proof that in abdominal operations post-operative chest complications are six times more frequent in smokers than in non smokers.

If the patient gives a history of chronic winter cough which improves during the warm weather he should if possible have his operation postponed until he is at his best. Chemo-therapy, penicillin, postural drainage etc. may be suggested in suitable cases e.g. the technique described by Palmer and Sellick in which postural drainage and physiotherapy are combined with inhalation of the bronchodilator isoprenaline can be employed.

Lost nasal catarrh and sinus infection should be sought out and treated while viscosity of sputum can often be reduced by the daily administration of potassium iodide 0.40 gr. for a few days before operation.

Breathing exercises should be started in the clinic and should be conducted if possible by the same personnel who will later be met in the ward after operation. Group therapy has a place here and classes of patients can be put through a suitable drill with advantage both to their physique and their morale.

2. **TEETH AND GUMS**—There is no longer an economic excuse for patients to come for operation with carious or infected or dangerously loose teeth. Patients so afflicted should be referred to the dentist for advice and treatment. If extractions have to be done time should be given for the gums to be well healed before operation.

3. **CARDIOVASCULAR SYSTEM**—The ability of the heart and vessels to withstand strain has an important bearing on the prognosis of anaesthesia and operation. Cardiac efficiency tests

*Moving the Patient continued*

For patients who have been in the Trendelenburg position during operation and in patients suffering from circulatory depression a trolley which can maintain a head-down tilt should be used to take the patient back to bed

- 4 Position in Bed** —The patient should lie in the semi prone position until his reflexes return This is maintained by a pillow between the bed and the chest the lower arm is placed behind the trunk the upper knee is flexed This helps to maintain a free airway by causing the tongue to fall away from the posterior pharyngeal wall it also helps to prevent aspiration of vomitus into the air passages

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Lost nasal catarrh and sinus infection should be sought out and treated while viscosity of sputum can often be reduced by the daily administration of potassium iodide 20-40 gr. for a few days before operation.  
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Cardiovascular System *continued*

such as those of Sabrasez and of Crampton can be carried out in the clinic if thought necessary

Organic heart disease may be suspected in patients under 40 if one or more of the following signs are present (a) Definite cardiac enlargement (b) Diastolic murmur (c) Hypertension (d) Arrhythmias such as auricular fibrillation or heart block (e) History of either anginal attacks or congestive heart failure

In older patients a history of increasing dyspnoea on exertion attacks of angina of effort or of congestive heart failure put the anaesthetist on his guard

Coronary disease may be suspected in patients with a history of angina of effort cardiac asthma or acute pulmonary oedema It may be present without signs of atheroma in other arteries It has been said (A R Gilchrist) that a patient who can take a brisk walk without retrosternal pain on a cold day probably has not got coronary disease

No non urgent operation should be performed on a patient within three months of an attack of coronary occlusion

Adequate treatment before operation will greatly improve the chances of certain cardiopaths of surviving their surgical ordeal successfully If patients with progressive angina of effort must be anaesthetized the daily administration of 240-480 mg of aminophylline for a few days pre operatively may be helpful

The anaesthetist will be forewarned if he is able to detect such conditions as (a) Recent coronary occlusion (b) Syphilitic aortic stenosis (c) Severe angina pectoris (d) Complete heart block with Stokes Adams attacks Such cases are grave risks and demand an impeccable anaesthetic technique

- 4 THE BLOOD—Secondary anaemia must be diagnosed and treated It is better to give iron pills in the out patient clinic than one or more transfusions in the ward The opportunity should be taken of sending a specimen of blood to the pathologist for hæmoglobin and urea estimations and blood grouping He will appreciate not having to do these investigations hurriedly in the few hours before the operation

Patients who do not respond to iron given by mouth often improve if it is given intravenously as saccharated iron oxide About 25 mg are necessary to raise the hæmoglobin by 1 per cent The initial dose is 50 mg which can later be increased Nausea vomiting abdominal pain and backache are some times seen after injection

- 5 NUTRITION—Patients who are dyspeptic tend to live on inadequate rations and may show evidences of hypoproteinæmia which may have serious consequences after operation The addition of protein supplements skimmed milk etc to the diet will help such patients Likewise vitamins can be prescribed if thought to be necessary

Obesity is always an enemy of both surgeon and anæsthetist. If treated properly by diet providing 1000 calories daily and such drugs as dextro-amphetamine sulphate 10 mg before breakfast and lunch excellent results may be obtained even in the few weeks at the anæsthetist's disposal.

- 6 **EMOTIONAL MAK-UP** —The patient's fears and anxieties can be discussed much more readily in the calm atmosphere of the clinic than in the ward. His emotional tone can be assessed so that premedication and type of anæsthetic can be tailor-made to fit his requirements. There are certain patients who are not suitable for techniques of regional analgesia and these can be sorted out in the clinic.

- 7 **PREVIOUS ANÆSTHETIC HISTORY** —A fuller discussion of this and its relationship to the proposed operation and anæsthetic can be undertaken in the clinic than in the ward. Unless there are very good reasons to the contrary a patient's wishes in regard to choice of anæsthetic and technique should be given sympathetic consideration.

The nares, sacral region, lumbar spine, arm and leg veins, abdominal wall, etc. should be examined and any abnormality noted.

Quite frequently the anæsthetist will need the assistance of his medical, radiological, pathological or laryngological colleagues and not the least of the benefits of such a clinic is gained by the anæsthetist himself in the close personal and clinical contact established there. By making himself as far as possible competent to run such clinics the anæsthetist has a great contribution to make to the recovery and rehabilitation of the surgical patient.

One other advantage of the thorough pre-operative investigation of the patient is the assurance and confidence that the anæsthetist feels should medico-legal proceedings be instituted by the patient after operation. It is the fate of those who toil at the lower employments of life to be exposed to censure without hope of praise, to be disgraced by miscarriage or punished for neglect where success would have been without applause and diligence without reward. (Dr Samuel Johnson.) Among these unhappy mortals is the administrator of anæsthetics and he must cover himself in every way possible.



## CHAPTER XXXI

## ANÆSTHESIA AND ANALGESIA IN LABOUR\*

## CHOICE OF ANÆSTHETIC

While it is held by many workers that in normal labour all that is necessary is complete mental and physical relaxation as advocated by Grantly Dick Read the majority of patients demand the aid of more material pain relieving techniques. There is a definite maternal death rate for which the anæsthetist must take some blame. This is associated with (1) Aspiration of vomitus (2) Hypotension associated with regional analgesia (3) Inadequate blood transfusion.

Vomiting is always a real danger during labour as the patient may not be suitably prepared while the gastric emptying time is delayed. In addition the rather light plane of anæsthesia required is often the cause of vomiting. It is wise to prevent ingestion of all solid food during labour relying on fluids, sweets, isotonic glucose etc. Before induction of anæsthesia a large stomach tube should be passed in all cases of doubt.

Severe cases of aspiration require careful tracheobronchial toilet, hydrocortisone and perhaps bronchoscopy. Milder cases may be treated by (a) The Trendelenburg position (b) Lightening anæsthesia to encourage coughing (c) Pharyngeal suction. As the trouble is mainly caused by irritative gastric contents atropine, oxygen, aminophylline, chemotherapy and antibiotics should be employed where necessary.

Every labour ward should be equipped with (1) A tipping table † (2) An efficient suction apparatus (3) Laryngoscope and endotracheal tubes (4) A transparent face mask (5) A bronchoscope (6) A tracheotomy set.

**Prevention of Vomiting during Anæsthesia in Labour**—Aspiration of gastric contents from the stomach is a real danger when a general anæsthetic is given to patients in labour because of the delayed emptying time of the stomach. Trouble may arise from (a) Gross obstruction by solid or liquid material (b) By an asthmatic and bronchospastic response due to the inhalation of irritating acid gastric contents (Mendelson's syndrome‡) (c) Or from bronchopneumonia and its later complications e.g. lung abscess or bronchiectasis. Regional analgesia avoids these dangers but may not always be convenient either to the patient or to one or other of her medical attendants.

For a fuller account of pain relief in labour see *The Practical Management of Pain in Labour* by W. D. Wylie, 1953, London, Lloyd-Luke. *The Relief of Pain in Childbirth* by W. C. W. Nixon and S. G. Ransom, 1951, London, Cassell & Co. *Inhalation Analgesia in Childbirth* by E. H. Seward and R. Bryce-Smith, 1957, Oxford, Blackwell. Discussion on Anæsthesia for Obstetrics, Dunnick, O. P., Steele, G. L. and others, *P. or R. Soc. Med.* 1947, 50, 547.

† Wylie, W. D., *Lancet* 1956, 1, 840. Glibbert, G. P., *Ibid.* 1955, 1, 901.

‡ Mendelson, C. L., *Amer. J. Obst. Gynec.* 1946, 52, 191.

Aspiration of stomach contents during general anaesthesia can be made less likely by —

- 1 Giving only fluid and semi solid material during labour
- 2 The insertion of a number 12 œsophageal (or number 20 Wangenstein) tube before induction of anaesthesia
- 3 Inducing anaesthesia on a bed which can rapidly be tilted head down \*
- 4 Using thiopentone ( 50–250 mg ) for induction and gas oxygen trilene or ether for maintenance †
- 5 Inducing anaesthesia in the lateral position Avoiding strapping face piece on to face using a face piece made of transparent material
- 6 Inducing anaesthesia with equal volumes of cyclopropane and oxygen with the head elevated to prevent regurgitation followed by the insertion of a cuffed endotracheal tube under suxamethonium (50 mg) ‡
- 7 Inducing anaesthesia with the head elevated by thiopentone (200–250 mg) and suxamethonium (50 mg) followed by the insertion of a cuffed endotracheal tube
- 8 Methods 6 and 7 should only be used by anaesthetists of some experience The tyro would perhaps do best with gas oxygen trilene ether with a little carbon dioxide in the early stages and using the lateral position The presence of a sucker would be an additional safety factor
- 9 Intravenous injection of gr  $\frac{1}{8}$  of apomorphine to induce vomiting When the stomach has thus been emptied atropine gr  $\frac{1}{8}$  is given slowly intravenously to combat the vagal effects sometimes seen after apomorphine This treatment does not markedly disturb the patient §

It is often convenient to use drugs with a continuous action during the first stage of labour and those with an intermittent action at the end of the first and during the second stage

The ideal anaesthetic should —

- a Produce efficient relief from pain with consciousness between pains and good co-operation from the patient
- b Not depress the respirations of the foetus
- c Not depress the uterus causing prolonged labour
- d Be non toxic
- e Be safe for mother and child

No agent at present in use fulfils all these conditions

Liquor amnii is sucked in and pushed out of the lungs by the action of the foetal diaphragm and intercostal muscles All anaesthetics and all analgesic agents other than bromide and chloral in reasonable dosage depress the foetal respiratory mechanism an effect made worse by any hypoxia of the mother during labour The placenta acts as no barrier to these agents There is definite evidence that hypoxia of the foetus during labour and of the baby at birth or shortly afterwards may be

Wyle W D *Lancet* 1956 1 840 Gibberd G F *Ibid* 1955 1 901

† Crawford J S *Brit J Anaesth* 1956 28 146 28 201

‡ Wyle W D *Proc R Soc Med* 1955 48 1089

§ Holmes J St *Ibid* 1957 50 556

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Wyl W D *Lancet* 1956 **1** 840 Gibberd G F *Ibid* 1955 **1** 901

† Cr wlo d J S *Br J A nth* 1956 **28** 146 28 201

‡ Wyl W D *Proc R Soc Med* 1955 **48** 1089

§ Holmes J M *Ibid* 1957 **50** 556

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† Wyllie, W. D. *Lancet* 1956, 1, 840. Gibberd, G. F. *Ibid.* 1955, 1, 905.

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† Wylie, W. D. *Lancet* 1956, 1, 840. Gibberd, G. F. *Ibid.* 1953, 1, 901.

‡ Mendelson, C. L. *Amer. J. Obst. Gynec.* 1946, 52, 191.

Because it is an amnesic it is sometimes combined with pethidine, barbiturates, etc. Has been known to cause oedema of uvula. It should not be given late in labour as then it may cause restlessness and lack of co-operation. It passes the placental barrier. One dose of morphine, scopolamine or pethidine and scopolamine is often useful at the beginning of labour.

**TWILIGHT SLEEP**—Morphine and scopolamine were used in 1902 by Gauss and von Steinbüchel of Frankfurt. To-day it is not very popular because of—

- a Its uncertain effects
- b Its tendency to cause prolonged labour
- c Its tendency to cause excitement and restlessness of the mother
- d Its tendency to produce foetal asphyxia
- e The requirement of skilled observers throughout the labour

On the other hand it is easy to give, has a rapid effect, is safe for the mother and is not costly.

**TECHNIQUE**—When labour has definitely started and is causing distress the first injection is given of morphine  $\frac{1}{4}$ – $\frac{1}{2}$  gr (10 to 16 mg) with scopolamine  $\frac{1}{16}$ – $\frac{1}{8}$  gr (0.4 to 0.6 mg). Omnopon can be substituted for the morphine. The patient under careful observation throughout labour is left to sleep in a darkened room and in 1–1½ hours scopolamine  $\frac{1}{16}$ – $\frac{1}{8}$  gr (0.15 mg) is injected and repeated when amnesia is receding as shown by the ability of the patient to recognize simple objects such as a safety pin or powder puff which have recently been shown to her. Injections average out about once every 1–1½ hours. The dosage must be individualized to suit each patient. Moderate restraint may be required with the pains but between them the patient should sleep. The method produces efficient amnesia in a reasonable percentage of cases.

Twilight sleep should not be commenced too early in labour nor in cases of primary uterine inertia because of the excessive doses which will be required. Thirst must be attended to, the bladder catheterized when necessary. During the second stage other agents should be used to control pain such as trilete or nitrous oxide.

- 4 **Barbiturates** (see also p. 64)—Pentobarbitone (nembutal) is most commonly employed but butobarbitone, sodium quinalbarbitone and sodium amytal have also been used. Barbiturates are less depressing to the foetal respiration than morphine but more so than rectal paraldehyde or ether-oil. The effects are uncertain and restlessness, drowsiness or delirium may result. The addition of caffeine reduces foetal respiratory depression and increases the mother's co-operation. The addition of alkali hastens the action of pentobarbitone. Reasonable doses of pentobarbitone sodium have no serious effect on the uterine contractions but are reported to have caused acute pulmonary oedema. Like other barbiturates pentobarbitone may be responsible for lowering the prothrombin level of the blood of both the mother and foetus. After the administration of barbiturates to the mother in labour the foetus may remain sluggish for a day or two after birth as the foetus is unable to detoxicate barbiturates as rapidly as the mother.



## Choice of Anæsthetic continued

followed by impaired cerebral function in later life \* The danger is increased with premature infants

**1 Simple Sedatives** — *Chloral hydrate* a soporific and mild analgesic is only a cardiac depressant when given in large doses. It is excreted by the kidneys with glycuronic acid. The syrup contains 10 gr in each drachm. A safe dose is 30 gr together with *potassium bromide* (30 gr) and perhaps *tincture of opium* (10–15 min). A view held by some pharmacologists but disputed by clinicians is that bromides only act after displacing chlorides from extracellular fluid so are useless in single doses. These drugs can be given early in labour well diluted to prevent vomiting and can be repeated. They are useful in the early stages of labour often allowing the patient to sleep between pains. *Glutethimide* (dorden) in 500 mg doses is also a useful sedative in labour †

**2 Morphine** (see also p 52) — Morphine was not much used in labour until Steinbuchel combined it with scopolamine in 1902 this was because of its depressant effect on foetal breathing. An injection of morphine  $\frac{1}{2}$  gr or heroin  $\frac{1}{8}$  gr is often very useful in labour and is more efficient if given before severe pain comes on. The maximal effect on pain relief is shown ninety minutes after intramuscular injection. The drug must be given with care in shocked exhausted or exsanguinated patients. The maximal depression of foetal respiration occurs about two hours after injection so it should not be given within 2½–3 hours of the expected time of delivery.

**EFFECT ON MOTHER** — (1) Elevates pain threshold (2) Alters attitude of patient to her pain producing relative indifference (3) Acts as a hypnotic

**EFFECT ON FÆTUS** — (1) Before labour — none which is harmful (2) During labour it severely depresses the foetal respiratory mechanism soon crossing the so-called barrier of the placenta — worse in the premature than in the normal foetus

**EFFECT ON PROGRESS OF LABOUR** — In small doses morphine increases the intervals between pains in larger doses it may depress contractions

*Amiphenazole* — A respiratory stimulant can be given to the mother (30 mg intramuscularly) shortly before the delivery of the child or directly into the umbilical vein (3 mg) in cases where morphine or pethidine has been injected into the mother at a rather late phase of labour. It reverses the respiratory depressant effects but not the analgesic effects of the sedatives morphine dromoran pethidine etc

*Nalorphine* — Has a similar use Dose 3 mg (see Chapter XIV)

**3 Scopolamine** (see also p 62) — (1) Does not increase pain threshold (2) Alters patient's reaction to pain by producing amnesia excitement and sometimes delirium resembling acute mania. Does not depress respiration of foetus nor affect the course of the labour

the most useful single drug employed in labour. First used in labour by Benthin in 1940. It does not result in lack of co-operation.

**PHARMACOLOGY** —It relieves pain having an action midway between morphine and codeine. A dose of 100 mg of pethidine is roughly equivalent to  $\frac{1}{4}$ – $\frac{1}{2}$  gr of morphine. It does not produce amnesia but causes sleepiness and drowsiness for about 2 hours. Side-effects are dizziness, faintness. Is somewhat uncertain in its action.

It reduces smooth muscle spasm by —

- a A direct papaverine like effect on the muscle fibres
- b An atropine-like depressant effect on the parasympathetic nerve-endings

Thus in labour it raises the pain threshold and reduces cervical spasm.

The blood pressure may be depressed if given intravenously unaltered if given intramuscularly. There is an increase in the incidence of vomiting and the patient may experience vertigo and tingling of the extremities. It can produce addiction.

As it depresses foetal respiration it should not be given within 3 hours of delivery. This depression is less marked than that due to morphine. About 10 per cent of infants require resuscitation. Analgesia is good in about 60 per cent of cases. There is no increase in the instrumental delivery rate. The patient usually sleeps during labour which is shortened because of more rapid cervical dilatation. It is very useful in cases with a rigid slowly dilating os. Sensitivity to the drug with symptoms of circulatory and respiratory depression has been described.

**DOSAGE** —Initial dose 100 mg when labour is well established. Should not be given too soon as then it may abolish pains. Usually given by intramuscular injection when effect comes on in 15 minutes and is maximal 1–1½ hours after injection in both mother and foetus (respiratory depression). If given by mouth it may cause vomiting. Additional similar doses are given when required e.g. every 2–3 hours. Maximum dosage for a midwife working alone is 200 mg in any one labour.

It has been combined with pentobarbitone and also with scopolamine to increase the amnesic effect. A well recommended technique is to inject intramuscularly pethidine 100 mg and scopolamine  $\frac{1}{16}$  gr when contractions are occurring regularly and when the os shows definite dilatation. Repeat doses are given if required but no pethidine should be injected within three hours of the expected delivery. The drug is of greatest use for first stage labour pains. The same drugs and doses well diluted in saline can be injected very slowly intravenously on one or two occasions. Its respiratory depressant effects on the infant can be prevented or cured by amiphenazole and also by nalorphine and levallorphan. The dose of nalorphine is 10–20 mg intravenously to the mother or 0.5 to 1 mg for the infant.

**Choice of Anæsthetic—Barbiturates continued**

The initial dose of pentobarbitone or quinalbarbitone is 3 gr given when labour is well established with the cervix dilated to two fingers. Additional doses of 1½ gr are given every 3-4 hours. It is unwise to exceed 9 gr in 12 hours.

Chloral hydrate can be combined with pentobarbitone 20 gr being given ½ hour after initial dose of pentobarbitone. Small additional doses are given when required with a safe maximum of chloral 120 gr and pentobarbitone 7½ gr in 12 hours.

Scopolamine ½ gr with ½ gr later if required has been added to pentobarbitone to increase its amnesic effect.

Very good results follow the combination (a) Quinalbarbitone (seconal) 100 mg by mouth (b) Pentobarbitone (nembutal) 100 mg intramuscularly with 0.6 mg of scopolamine (c) Pethidine 100 mg intramuscularly—all given when active labour starts. Scopolamine 0.2 mg can be repeated two hourly.

- 5 Paraldehyde** (*see also p 225*)—First used in obstetrics by Rosenfeld and Davidoff in 1932. It raises the pain threshold, produces hypnosis and sometimes excitement. Elimination is slow, only a small portion being via the lungs. Much is destroyed in the body. It depresses uterine activity but is not a powerful depressor of fetal respiratory activity. The usual dose is 60 min per stone body weight with a maximum of 1 oz. Each 60 min is well shaken with 1 ½ oz of oil or saline and is instilled into the rectum via a catheter so placed that its tip is above the presenting part. It is given just after a pain with the patient on her left side. The first dose is given when labour is definitely established with the patient experiencing regular pains. In suitable cases it can be preceded by pentobarbitone, morphine or scopolamine. The patient remains drowsy for 3-4 hours, labour may be retarded and restlessness caused. The infant may be mildly narcotized and may remain drowsy for some hours but respiration is almost always started without difficulty. In long labours second injections can be given if necessary.

The method is very safe.

- 6 Bromethol (Avertin)** (*see also p 223*)—The usual dose is 75 mg per kilo after deducting 6 kilos for the weight of the uterus and its contents. The effect lasts 1½ hours and may result in prolonged labour and fetal apnoea. After bromethol the patient takes light general anæsthesia e.g. gas and oxygen very well. This method is not recommended as an anæsthetic in labour but has its place in the treatment of eclampsia.

- 7 Pethidine** (*see also p 57*)—Known also as dolantin, dolantal, isompercan, mependine hydrochloride and demerol, it is the hydrochloride of the ethyl ester of 1-methyl-4-phenyl piperidine-4-carboxylic acid and was synthesized in 1939 by Schaumann and Eisler.

If certain rules propounded by the Central Midwives Board are observed it can be used by midwives acting alone. Probably

the most useful single drug employed in labour. First used in labour by Benthin in 1940. It does not result in lack of co-operation.

**PHARMACOLOGY** — It relieves pain having an action midway between morphine and codeine. A dose of 100 mg of pethidine is roughly equivalent to  $\frac{1}{4}$ – $\frac{1}{2}$  gr of morphine. It does not produce amnesia but causes sleepiness and drowsiness for about 2 hours. Side-effects are dizziness, faintness. Is somewhat uncertain in its action.

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**DOSAGE** — Initial dose 100 mg when labour is well established. Should not be given too soon as then it may abolish pains. Usually given by intramuscular injection when effect comes on in 15 minutes and is maximal 1–1½ hours after injection in both mother and foetus (respiratory depression). If given by mouth it may cause vomiting. Additional similar doses are given when required e.g. every 2–3 hours. Maximum dosage for a midwife working alone is 200 mg in any one labour.

It has been combined with pentobarbitone and also with scopolamine to increase the amnesic effect. A well recommended technique is to inject intramuscularly pethidine 100 mg and scopolamine  $\frac{1}{16}$  gr when contractions are occurring regularly and when the os shows definite dilatation. Repeat doses are given if required but no pethidine should be injected within three hours of the expected delivery. The drug is of greatest use for first stage labour pains. The same drugs and doses well diluted in saline can be injected very slowly intravenously on one or two occasions. Its respiratory depressant effects on the infant can be prevented or cured by amiphenazole and also by nalorphine and levallorphan. The dose of nalorphine is 10–0 mg intravenously to the mother or 0.5 to 1 mg for the infant.

Choice of Anæsthetic *continued*

- 8 Nisental (Alphaprodine)** (*see also p. 58*)—This drug acts in a similar way to pethidine but differs from it (1) by acting more rapidly (2) by lasting a shorter time (3) by being less potent. It may cause mild respiratory depression in the infant but has few unpleasant side effects in the mother. It should be withheld until labour pains are occurring regularly and are well established. The initial dose is 40–80 mg and this causes a mild sleepy euphoria lasting two to three hours. It combines well with scopolamine.

**9 The Phenothiazine Derivatives—**

- a CHLORPROMAZINE**—Opinions differ as to the utility of this drug in labour. It undoubtedly relieves nausea and vomiting both before and during labour and would appear to be relatively harmless to the child. It may however cause delay in labour. The slow intravenous injection of pethidine 150 mg with chlorpromazine 50 mg 10 to 15 minutes before forceps delivery has given good results in regard to both mother and infant\*. It can be usefully combined with local infiltration of the perineum and internal pudendal nerve block.

- b PROMETHAZINE**—Used with pethidine—25 mg of promethazine and 50 mg of pethidine intramuscularly repeated as required the drugs form a useful sedative in labour and cause no harmful side effects to mother or child if used in reasonable amounts. The mother becomes sleepy and contented but can be roused and then becomes co-operative.

- 10 Rectal Ether oil** (*see also p. 227*)—This method was introduced into obstetrics in 1913 by Gwathmey who used ether-oil magnesium sulphate and morphine. The original method has been altered and to-day McCormick's modification is used in the United States but not often in Britain. It consists of oral pentobarbitone (nembutal) followed by rectal ether-oil with or without the addition of rectal paraldehyde.

It is safe for mother and child, easy to administer in the home and fairly efficient as an analgesic giving good results in 80 per cent of cases. Local disease of the rectum is the only absolute contra-indication. Analgesia not anæsthesia results from the rectal injection so that the airway is not interfered with through muscular relaxation of the tongue and pharynx. Labour is not prolonged, the forceps rate is not increased and the number of stillbirths is not influenced.

The patient requires careful supervision throughout labour.

**TECHNIQUE**—At the commencement of labour in primigravida an enema of sod bicarb 60 gr to the pint of water is given. When the patient is showing discomfort pentobarbitone 3–4½ gr is taken by mouth and when further genuine pain is complained of pentobarbitone 3 gr or morphine ½–1 gr is given. When the effects of this wear off the following enema is injected into the rectum from a syringe and catheter under pressure.

high above the presenting part ether  $\frac{1}{2}$  oz (75 ml) olive oil or liquid paraffin  $1\frac{1}{2}$  oz (45 ml). In suitable cases *formaldehyde* 10 min (75 ml) is added to the ether-oil mixture. The maximum effect comes on in half an hour and analgesia lasts 2-6 hours. A similar rectal injection is repeated when needed—on an average of 2-3 times in primipara.

In this as in all the foregoing methods light general anæsthesia may be required during the birth of the baby. The patients take general anæsthesia well and usually require only small amounts. Care is required as in all cases of labour to see that the bladder is kept empty and that fluids and glucose are given. Close supervision is necessary.

- 11 Intermittent Chloroform Anæsthesia and Analgesia** (*see also* p. 103) —Chloroform is relatively safe in labour only because it is welcomed and not feared, hence ventricular fibrillation is seen less often than in other patients.

John Snow employed Sir James Young Simpson's technique of light chloroform analgesia during each pain when he administered it to Queen Victoria in 1853 at the birth of Prince Leopold—*anesthésie à la reine*.

Snow described his technique in his book *On Chloroform and Other Anæsthetics* (1858) as follows. It is desirable to give the chloroform very gently at first increasing the quantity a little with each pain if the patient is not relieved. The practitioner easily finds with a little attention the quantity of vapour it is desirable to give at any stage of the labour and in each particular case his object being to relieve the patient without diminishing the strength of the uterine contractions and the auxiliary action of the respiratory muscles or with diminishing it as little as possible. At first it is generally necessary to repeat the chloroform at the beginning of each pain but after a little time it commonly happens that sufficient effect has been produced to get the patient over one or two uterine contractions without suffering before it is resumed. Complete anæsthesia is never induced in midwifery unless in some cases of operative delivery. No better advice than this could be followed with chloroform or with any other method of intermittent analgesia e.g. trilene gas and air etc.

Chloroform can be given on a mask from brisettes—small glass capsules containing 20 min of chloroform which are broken on to a mask and give relief for two or three pains from Simpson's inhaler from Christie Brown's inhaler or from Junker's bottle or Mennell's modification of Junker's bottle. With the last apparatus the patient compresses a bulb which blows air through chloroform she uses it when she needs it and cannot produce true third stage anæsthesia as the vapour is too weak and intermittent. Mennell's bottle cannot be connected up wrongly cannot spill and cannot be overfilled. This method is still very popular in domiciliary midwifery. Chloroform can also be given from an anæsthetic machine using a Rowbotham bottle for its vaporization. Analgesia is brought

## Choice of Anæsthetic continued

**8 Nisentil (Alphaprodine)** (*see also p 58*) — This drug acts in a similar way to pethidine but differs from it (1) by acting more rapidly (2) by lasting a shorter time (3) by being less potent. It may cause mild respiratory depression in the infant but has few unpleasant side effects in the mother. It should be withheld until labour pains are occurring regularly and are well established. The initial dose is 40–80 mg and this causes a mild sleepy euphoria lasting two to three hours. It combines well with scopolamine.

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- 12 Intermittent Trilene** (see also p. 110) —The first apparatus for the self administration of trilene and air was introduced in 1943 (Freedman). This is a useful drug in labour. It gives better results in nervous highly strung women than nitrous oxide and in patients whose labours are likely to be over within six hours. If trilene is given intermittently for longer than six hours the patient may show signs of drowsiness and lack of co-operation. Such a state of affairs can be controlled by reducing the vapour strength of trilene to 0.3 or 0.4 per cent and by giving a dose of pethidine 50 mg. Vapour of 0.5 per cent concentration and

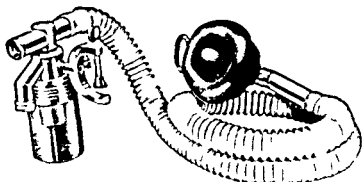


Fig. 70.—The Freedman's (Woodfield Davies modification) (British Oxygen Co. Ltd.)

less does not materially influence uterine contractions. Like other anæsthetic agents it rapidly enters the fetal circulation but has not been proved definitely to harm the fetus. In some animals its concentration in fetal blood soon exceeds that in maternal blood. It is possible that drugs used to augment action of trilene such as pethidine or morphine may be at least partly responsible for fetal respiratory depression. Trilene-air as compared with nitrous oxide-air causes a slight rise in systolic blood pressure during second stage pains. It has been given from a Rowbotham's chloroform bottle (Fig. 69) attached to a gas-air or gas-oxygen machine from Marrett's draw over apparatus from Freedman's inhaler (Fig. 70). This apparatus which cannot be overfilled is clamped to the bed and can be used by the patient unassisted. It consists of face mask, corrugated tubing, and bottle and as a safety device a hole is provided over which the patient places her finger. If this slips due to too deep a level of analgesia air is inhaled instead of trilene vapour. It delivers about 0.65 per cent trilene in air (by weight) but this vapour strength varies with environmental temperature, agitation of the bottle, etc. so that the authorities are not willing to place the apparatus in the hands of midwives working alone. It has also been used in an Oxford vaporizer when 25 per cent on the ether scale gives a concentration of trilene in air of approximately 1.5



*Intermittent Chloroform Anæsthesia and Analgesia continued*

about by the inhalation of 0.1-0.3 per cent chloroform vapour in air

Chloroform analgesia will retard labour even given by Snow's technique it may predispose to post partum hæmorrhage and may injure the liver. Chloroform will relax a labouring uterus better than any other drug.

Protection of the liver from the effects of chloroform include

- (1) A diet rich in carbohydrate poor in fat (2) Addition of

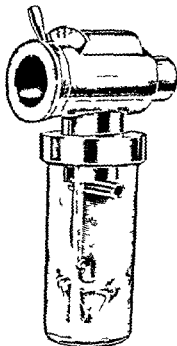


Fig. 63.—Rebreather bottle (British Oxygen Co. Ltd.)

oxygen to the inhaled gases (3) Methionine and cystine even if given within four hours of the end of the anæsthesia. The drug is positively contra indicated in patients with depleted liver glycogen e.g. in toxæmia or starvation (persistent vomiting).

The sudden inhalation of a strong concentration may be dangerous and may cause severe cardiac depression. Instant artificial respiration is indicated.

The Royal College of Obstetricians and Gynaecologists is of the opinion that a midwife working alone should not use chloroform.

must be inspected periodically by the manufacturer to test its accuracy and that midwives using it must be properly trained in its use.

- 13 **Gas air and Gas oxygen**—Nitrous oxide-air (4:24) is a better analgesic mixture than nitrous oxide-air (5:5). The latter mixture if used over a long period of time cannot be pronounced entirely guiltless in the matter of fetal hypoxia. Nitrous oxide does not reduce labour pains. Gas and oxygen cannot be administered by a midwife working entirely on her own. Inhalation of 10-11 per cent oxygen as in a Minnitt machine reduces the oxygen tension in maternal blood to about 40 mm Hg (normal 90) in 30 seconds so that even intermittent gas and air with a defective placental circulation may cause intra uterine asphyxia. See also Chapter VIII.
- 14 **Ether Cyclopropane and Vinesthene** These are usually given from a gas machine during the birth of the child. Vomiting during induction or maintenance is a frequent complication and may be a danger. All of these agents depress the foetal respiratory centre before the mother reaches the stage of surgical anaesthesia and retard labour if pushed. Light cyclopropane is probably the least harmful in this respect. Plenty of oxygen should be given to the mother before the cord is cut and hypoxia must be avoided throughout the administration. Premature infants stand general anaesthesia badly.
- Cyclopropane is without effect on the liver and kidneys and so is useful in toxemia. It produces apnoea in the baby proportional to the depth and duration of anaesthesia. It does not predispose to post partum haemorrhage and enables good retraction to take place in the uterus after delivery. If posterior pituitary extract is given to a patient receiving cyclopropane death from ventricular fibrillation or coronary constriction may occur: this applies to its pressor but not its oxytoxic fraction.
- Ether* can be given by the open drop method after induction:
  - (1) By *trilene* from an inhaler—especially the Marrett or Hyatt both of which are capable of delivering a relatively high vapour strength.
  - (2) By *vinesthene*.
  - (3) By *ethyl chloride*.
 It can also be given from an anaesthetic machine and thio-pentone may make the induction not only more pleasant but smoother and therefore safer.
- Ether air is far less explosive than ether-oxygen (explosive range in air 1.85-36.5 per cent; explosive range in oxygen 2.1-8.2 per cent). Before ether air is administered in a bedroom gas and electric fires should be put out and a coal fire damped down. Ether vapour being heavier than air will accumulate on the floor.
- Hal than* tends to depress uterine tone in the deeper planes of anaesthesia.
- 15 **Thiopentone**—This should be used sparingly because of its depressant effect on the foetal respiratory centre. It would appear that the placenta forms no barrier to thiopentone and that the foetal blood level of the drug is at a maximum at the onset

*Intermittent Trilene continued*

per cent the Cyprane inhaler which delivers a variable percentage vapour in air of 0.22 per cent to 0.54 per cent the Hyatt inhaler similar to the Cyprane but with a greater range of percentage concentrations. The most accurate and elaborate apparatus designed for self administration of trilene vapour is the Emotril Automatic Inhaler (Epstein Macintosh Oxford TRILENE) which delivers 0.5 per cent trilene in air and is compensated for changes

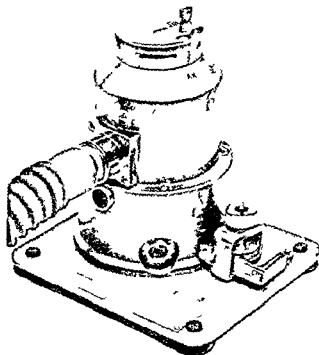


FIG. 71—The Epstein Macintosh Oxford TRILENE (Cyprane Ltd.)

in environmental temperature. It will also give a weaker vapour (0.35 per cent) should the patient become too drowsy with the higher concentration. Other temperature compensated appliances include the Tecota Inhaler (F. & T. Temperature Compensated Trilene Air), the Airline Inhaler and the Burns Benson Inhaler. The Central Midwives Board has approved the following apparatus for the administration of trilene by midwives: the Tecota Mark 1 (Cyprane Ltd.) and the Emotril Automatic Inhaler (Medical and Industrial Equipment Ltd.). They stipulate that such apparatus

must be injected perfectly by the manufacturer if accuracy and that involves using the instrument perfectly trained in the use.

- 13 Gas air and Gas oxygen**—Nitrous oxide (5-25) is a better analgesic mixture than nitrous oxide air (5-5). The latter mixture if used over a long period of time cannot be pronounced entirely guiltless in the matter of foetal hypoxia. Nitrous oxide does not reduce foetal pain. Gas and oxygen cannot be administered by a midwife working entirely on her own. Inhalation of 10-11 per cent oxygen as in a Minnitt machine reduces the oxygen tension in maternal blood to about 40 mm Hg (normal 90) in 10 seconds so that even intermittent gas and air with a defective placental circulation may cause intra-uterine asphyxia. See also Chapter VIII.

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Cyclopropane is without effect on the liver and kidneys and is useful in toxæmia. It produces apnoea in the baby proportional to the depth and duration of anaesthesia. It does not predispose to post partum haemorrhage and enables good retraction to take place in the uterus after delivery. If posterior pituitary extract is given to a patient receiving cyclopropane death from ventricular fibrillation or coronary constriction may occur. This applies to its pressor but not its oxytocic fraction.

*Ether* can be given by the open drop method after induction. (1) By *trilene* from an inhaler—especially the Marrett or Hyatt both of which are capable of delivering a relatively high vapour strength. (2) By *vinesthene*. (3) By *ethyl chloride*. It can also be given from an anaesthetic machine and thiopentone may make the induction not only more pleasant but smoother and therefore safer.

Ether air is far less explosive than ether-oxygen (explosive range in air 1.85-36.5 per cent explosive range in oxygen 2.1-82 per cent). Before ether air is administered in a bedroom gas and electric fires should be put out and a coal fire damped down. Ether vapour being heavier than air will accumulate on the floor.

*Halothane* tends to depress uterine tone in the deeper planes of anaesthesia.

- 15 Thiopentone**—This should be used sparingly because of its depressant effect on the foetal respiratory centre. It would appear that the placenta forms no barrier to thiopentone and that the foetal blood level of the drug is at a maximum at the onset

*Thiopentone continued*

of anaesthesia and thereafter falls \* A dose of 200-250 mg used for induction of anaesthesia is unlikely to have a serious depressive effect on the infant's breathing capacity but further doses are probably undesirable

For external version thiopentone may not give adequate relaxation. It is often undesirable for forceps delivery except as an inducing agent. If however a quick working obstetrician can promise a rapid delivery then 0.5 g together with gas-oxygen and perhaps *suxamethonium* can be used for episiotomy and outlet forceps. It is unsuitable for complicated cases and premature labours.

- 16 Subarachnoid Block** —(See also Chapter VIII) Can be used for mid or low forceps extraction or as a caudal procedure. For normal delivery or for outlet forceps with episiotomy block should extend to S1.

This can be obtained if the patient is placed in the lateral position on a level delivery bed or table. In such a position because the width of the hips is greater than that of the shoulders in most women there is a slight head-down tilt of the vertebral canal. Hyperbaric nupercaine (1-200 with 6 per cent glucose) 0.8 ml is injected between L4 and L5 and the patient immediately turned to the supine position with hips and knees flexed. The onset of analgesia takes about five minutes.

For high forceps or intra uterine manipulation block should reach T11. This will require the injection of 1-1.2 ml of hyperbaric nupercaine and will abolish the traction pain associated with a high forceps delivery which will result if the sacral nerves alone are blocked.

Other techniques for performing sacral (saddle) block are —

- 1 Injection into the sitting patient's subarachnoid space (L3-4 or L4-5) of —

a Nupercaine hyperbaric 0.5 ml with 0.5 ml of cerebrospinal fluid

b 30-50 mg of procaine in 1 ml of 5 per cent dextrose

c 4 mg of amethocaine in 1 ml of 5 per cent dextrose

The patient remains sitting for thirty seconds and then lies flat on her back with a pillow under her shoulders.

- 2 In the U.S.A. a favourite technique for performing saddle block is to inject into the sitting patient between L4 and L5 0.2 ml of 1 per cent pontocaine (amethocaine) solution i.e. 2 mg and 1 ml of 10 per cent glucose. The addition of 0.25 to 0.5 ml of 1-1000 adrenaline solution to these solutions will prolong the duration of block and slightly increase its extent because of the extra volume of injected solution. Perineal analgesia removes the bearing down reflex but saddle block does not greatly delay normal labour if when the cervix is fully dilated the patient is encouraged to bear down during the pains and if fundal pressure is applied

If used in congestive heart failure to help spare the mother the exertion of pushing out the baby subarachnoid injection should be made immediately the cervix is fully dilated

Advantages of subarachnoid block (1) No foetal respiratory depression (2) Excellent relaxation of pelvic floor muscles (3) Absence of aspiration of stomach contents and risk of asphyxia Mendelsohn's syndrome pneumonia etc (4) Delivery of patient while she is conscious

With all forms of regional block blood pressure fall must be avoided because of the risk of foetal hypoxia and to prevent this some workers give methamphetamine or methoxamine as a routine before the injection Pressure should not be allowed to fall below 90 mm Hg Intravenous injection of ergometrine soon after a patient has received a vasopressor may cause dangerous hypertension or even apoplexy

The method is not suitable if the head is high or if shock is present e.g. in cases of failed forceps The post partum uterus contracts well Post-operative headaches are rather frequent but their incidence can be reduced if a very fine needle preferably with a conical point and lateral eye is used \*

**17 Paravertebral Block.**—Paravertebral block of D 11 and D 12 on each side will abolish the pain of uterine contraction but not that due to cervical or perineal stretching and so has been used for giving relief from first stage pains The visceral afferent fibres must be blocked as they run in the white rami mixed spinal nerve and posterior root First used by J G P Cleland of Oregon in 1933 Supercaine 1-1500 or amethocaine 0.2 per cent is used 10 ml being injected into each nerve The effect lasts 2-3 hours

For technique see Chapter XVIII

**18 Extradural Sacral Block—Continuous Caudal Block**—(See also Chapter XVIII) Whatever the merits or demerits of continuous caudal block throughout labour there is no doubt about the excellence of a single injection given for forceps delivery In very obese patients Alvarez recommends that the middle finger of the left hand should be inserted into the rectum after the needle has pierced the skin to facilitate location of the sacral hiatus and canal Re sterilization of the hands of course follows before injections are commenced Continuous caudal block is relatively safe for mother and child gives superlative relaxation of the lower birth canal and gives good analgesia Xylocaine 1 per cent solution or metycaine 1½ per cent in Ringer's solution 20-30 ml are the recommended agents and doses Should continuation of the analgesia be decided upon topping up doses of 20 ml can be given when necessary The method should only be used when every means of resuscitation is to hand It is not without its disadvantages and is accompanied by a high forceps rate and increased frequency of anomalies of rotation e.g. persistent occipito-posterior position and mid transverse arrest of the head The third stage is short and

*Thiopentone continued*

of anaesthesia and thereafter falls \* A dose of 200-250 mg used for induction of anaesthesia is unlikely to have a serious depressant effect on the infant's breathing capacity but further doses are probably undesirable

For external version thiopentone may not give adequate relaxation. It is often undesirable for forceps delivery except as an inducing agent. If however a quick working obstetrician can promise a rapid delivery then 0.25 g together with gas-oxygen and perhaps *suxamethonium* can be used for episiotomy and outlet forceps. It is unsuitable for complicated cases and premature labours.

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  - c. 4 mg of amethocaine in ml of 5 per cent dextrose

The patient remains sitting for thirty seconds and then lies flat on her back with a pillow under her shoulders.

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Cappe B E. and Deutsch E V *Anæsthesiol* 57 1953 14 398 also Harris, L. M and Harmel M H., *Ibid* 1953 14 390



**Thiopentone continued**

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The patient remains sitting for thirty seconds and then lies flat on her back with a pillow under her shoulders.

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that the pudendal nerve can be blocked as it enters Alcock's canal at the level of the ischial spine or its two branches the perineal nerve and the dorsal nerve of the clitoris as they leave the canal. The needle is guided by a finger of the left hand in the vagina. Some solution should be placed posteriorly to the tuberosity of the ischium to block the perineal branch of the posterior cutaneous nerve of the thigh. From the same wheel the needle should be inserted subcutaneously in an anterior direction towards the symphysis pubis to block the ilio-inguinal nerve while lastly some more solution is injected from the wheel towards the sphincter of the anus to reinforce the block of the inferior haemorrhoidal nerve. Total volume of solution required is 70-80 ml. Adrenaline may inhibit labour pains and should not be used.

If the head is not yet distending the perineum the transvaginal route for infiltrating the pudendal nerves may be preferable to the transperineal route.

**b FOR EPISIOTOMY**—Infiltration is made between the skin and the mucosa of the vagina in the line of the proposed incision.

**c FOR REPAIR OF LACERATIONS**—A swab soaked in 2 per cent xylocaine 1-1000 amethocaine or 5 per cent metycaine is placed in the raw area of the tear to effect surface analgesia. After a few minutes  $\frac{1}{2}$  per cent solution of procaine is infiltrated. (a) In a plane parallel to the perineal skin. (b) In a plane parallel to the vaginal mucosa. In each case the needle is inserted from the raw area of the laceration.

**21 Presacral or Parasacral Block**—This involves block of the anterior primary divisions of the sacral nerves of the sacrococcygeal plexus and of the autonomic fibres in relation to the anterior surface of the sacrum. It is rarely done.

**INDICATION**—Forceps delivery, manual rotation of occipito-posterior positions with forceps delivery, breech extractions. Difficult cases of breech extractions requiring the hand to be inserted high into the uterus will require general anaesthesia in addition. A marked feature of the block is the good relaxation produced. Analgesia does not last as long as in extradural sacral (caudal) block. This method is not recommended.

**TECHNIQUE**—With the patient in the lithotomy position the sacrococcygeal joint is identified by deep palpation. Two wheels are raised each 2 cm from the midline at the level of the joint.

**1** A long needle is inserted through a wheel and makes contact with the edge of the last sacral vertebra. It is advanced parallel to the sagittal plane of the body along the anterior surface of the sacrum for about 7 cm when it will strike bone in the region of the second sacral foramen. As the needle is slowly withdrawn 60-70 ml of 0.5 per cent procaine with adrenaline are deposited between the second sacral vertebra and the sacrococcygeal joint.

**Extradural Sacral Block—Continuous Caudal Block** *continued*

post partum blood loss minimal The method is useful in uterine inertia and in cervical dystocia

- 19 Extradural Lumbar Block**—This was first used in obstetrics by Graffognino and Seyler in 1938 and as a continuous technique by Flowers Hellman and Hingson in 1949 Injection is between L 1 and L 2 or L 2 and L 3 with the patient on her side or sitting The recommended dose of 1 per cent xylocaine is 5–70 ml the smaller amount blocking the first stage pains (T 11 and 12) the larger amount the second stage (S 2 3 and 4) The continuous technique using a plastic catheter is satisfactory in labour For the second stage 15 ml of solution injected at the L 4–5 inter space with the patient sitting up—one injection—is satisfactory

- 20 Pudendal Nerve Block and Local Infiltration \***—This may be used for —

- a Normal delivery
  - b Episiotomy
  - c Outlet forceps
  - d Repair of laceration
- Indications may include foetal distress delayed second stage assisted breach delivery and multiple pregnancy

The hand cannot be inserted into the vagina without causing discomfort and good relaxation for intra uterine manipulation is not provided by this technique The method can be used for well over half the cases of forceps delivery including some mid forceps extractions \*

Xylocaine 0.5 per cent procaine 1 per cent or amethocaine or nupercaine 1–2000 can be used The addition of 150 turbidity reducing units of hyaluronidase to 30 ml of analgesic solution aids efficiency of the infiltration

**ANATOMY OF PUDENDAL NERVE**—This comes from the anterior divisions of the second third and fourth sacral nerves via the pudendal plexus It passes through the greater sacro sciatic foramen crosses the spine of the ischium medial to the internal pudendal vessels and enters the pelvis through the lesser sacrosclatic foramen With the pudendal vessels it passes upwards and forwards in Alcock's canal a tunnel in the fascia on the outer wall of the ischio-rectal fossa gives off an inferior hæmorrhoidal branch and finally divides into the perineal nerve and the dorsal nerve of the clitoris

- a **NORMAL DELIVERY**—Injections are commenced when the head is appearing at the vulva but before it distends the perineum A 12-cm needle is required for the deep injections The posterior part of the vulva must be infiltrated including the levator ani muscles and the perineum Injection is made down the posterior edge of each labium across the fourchette and between the vaginal wall and the rectum Injection should also be made into each ischio-rectal fossa to a depth of 5 cm from wheals midway between the anus and the tuberosity of each ischium so

that the pudendal nerve can be blocked as it enters Alcock's canal at the level of the ischial spine or its two branches the perineal nerve and the dorsal nerve of the clitoris as they leave the canal. The needle is guided by a finger of the left hand in the vagina. Some solution should be placed posteriorly to the tuberosity of the ischium to block the perineal branch of the posterior cutaneous nerve of the thigh. From the same wheal the needle should be inserted subcutaneously in an anterior direction towards the symphysis pubis to block the ilio inguinal nerve while lastly some more solution is injected from the wheal towards the sphincter of the anus to reinforce the block of the inferior haemorrhoidal nerve. Total volume of solution required is 70-80 ml. Adrenaline may inhibit labour pains and should not be used.

If the head is not yet distending the perineum the transvaginal route for infiltrating the pudendal nerves may be preferable to the transperineal route.

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- a **NORMAL DELIVERY**—Injections are commenced when the head is appearing at the vulva but before it distends the perineum A 1.5-cm. needle is required for the deep injections The posterior part of the vulva must be infiltrated including the levator ani muscles and the perineum Injection is made down the posterior edge of each labium across the fourchette and between the vaginal wall and the rectum Injection should also be made into each ischio-rectal fossa to a depth of 5 cm. from wheals midway between the anus and the tuberosity of each ischium so

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**Presacral or Parasacral Block** *continued*

2 The needle is again inserted at an angle of  $15^{\circ}$  to its former track still keeping in the sagittal plane this time for about 10 cm when the sacrum should again be struck in the region of the first sacral foramen and 20-30 ml of solution are injected in this area

3 Finally 10 ml of solution are deposited between the rectum and the coccyx The same procedure is repeated on the other side The empty rectum recedes before the needle point and is unlikely to be perforated Some workers keep the index finger in the rectum throughout the injections

Presacral block usually abolishes labour pains for 15-30 minutes but they come on again

In some cases local infiltration of the perineum is required in addition to ensure a painless delivery The third stage of labour proceeds normally

**22 Muscle Relaxants**—These agents are used in labour (1) To relax the perineum and abdominal wall (2) To aid endotracheal intubation There is evidence that gallamine enters the foetal circulation more readily than *d* tubocurarine or decamethonium and the agent should therefore be used only in small doses in labour Clinical doses of the remaining relaxants will probably do the infant no harm

The combination of nitrous oxide and oxygen anaesthesia with decamethonium (3-5 mg) or suxamethonium (30-60 mg) has given good results in cases of forceps delivery delivery of the aftercoming head episiotomy and repair of the perineum These drugs do not appear to cross the placental barrier in significant amounts They can also be used to prevent precipitate labour

**Anaesthesia for External Version**—After atropine gr  $\frac{1}{4}$  has been given and a suitable interval has elapsed anaesthesia is induced with thiopentone—0.25-0.5 g gallamine is then injected in serial doses the amounts required varying from 60 to 160 mg and gas with plenty of oxygen is gently insufflated In the rare case in which uterine tone prevents easy version ether is added to the gases until the uterus is fully relaxed The anaesthetist must wait until normal respiration has returned employing assisted respiration or neostigmine Thiopentone followed by suxamethonium will also give good results in the majority of cases Old fashioned obstetricians still have a liking for the efficient but highly questionable chloroform for these manipulations

**Anaesthesia for Internal Version**—If the obstetrician requires a well relaxed uterus high into which his hand and arm must be introduced ether is the best anaesthetic

**Anaesthesia for Forceps Delivery**—An efficient anaesthetic for forceps delivery should produce (1) Adequate relaxation of the muscles of the pelvic floor and perineum for the application of forceps (2) Minimal depression of labour pains (3) Minimal depression of post partum uterine contractions (4)

depression of the foetal respiration. The method chosen will depend on the environment, the experience of the anaesthetist and the facilities available. In domiciliary work any form of light general anaesthesia is suitable. A safe and relatively pleasant sequence is minimal thiopentone (50 or 75 mg) open veins, then (or ethyl chloride) followed by open ether. Thiopentone alone (50-75 mg) is suitable for a rapid outlet forceps extraction but not for one likely to take time.

In hospital low spinal analgesia has much to be said in its favour as it removes the risk of aspiration of gastric contents. Extra dural sacral block is excellent if time is available.

Gas oxygen trilene with or without minimal relaxant is probably the most popular technique to-day. A large stomach tube should be available and used frequently.

#### **Anaesthesia for Breech Delivery \*—**

- 1 In assisted breech delivery good oxygenation and a smooth induction at the right time are necessary. In the first and early second stage pethidine 100-150 mg and gas oxygen if necessary. When the presenting part appears an episiotomy can be done under infiltration analgesia and anaesthesia is not induced until the scapulae are delivered. It must then be rapid thiopentone (250 mg) with gas and oxygen or cyclopropane can be used for induction.

For breech extraction there is no hurry to induce anaesthesia which can follow the usually accepted techniques. Good relaxation may be required and for this maintenance with ether is often suitable.

**Anaesthesia for the Normal Delivery**—The following have been found to be helpful: (1) Encouragement of muscular relaxation. (2) Congenial company in early stage to maintain morale. (3) Rubbing of lumbar and sacral regions during early pains. (4) When pains become regular and distressing and os dilated to admit three fingers pethidine 100-150 mg injected intramuscularly. (5) If sleep is indicated chloral hydrate 30 gr can be given with the pethidine. (6) Late first stage is very painful and intermittent gas air may be necessary at this time. (7) With onset of second stage patient is encouraged to push and this makes the pains easier to bear. (8) When the head distends the perineum gas-air or trilene may be required and towards the end may need to be given (not self administered) almost continuously. Some patients will require full anaesthesia for the actual delivery and in domiciliary practice this may usefully be given by the doctor while the midwife delivers the patient.

**Anaesthesia for Retained Placenta.**—The patient may be shocked and a retraction ring may form an obstruction. Light thiopentone, light trilene and ether, gas oxygen ether—all are satisfactory. Inhalation of one or two capsules of amyl nitrite may relax a retraction ring if it does not deeper anaesthesia will be necessary. This drug may also relax a contraction ring earlier in labour.

**Anæsthesia in Abnormal Labour**—If labour is prolonged sleep at the proper time is necessary. When the cervix is slow in dilating or when a trial labour is under way larger than average doses of analgesics may be required. In severe anæmia or cardiac disease gas-air should be replaced by trilene vapour. Chloroform is contra indicated in toxæmia and in diabetes. In premature labour or where there is foetal distress sedatives should be withheld and gas-air should not be given. In foetal distress oxygen should be given to the mother and if forceps delivery is decided upon spinal or caudal analgesia may be safer for the baby than general anæsthesia. If the baby is dead or moribund then heavy sedation and adequate anæsthetic need not be withheld. Chloroform is the best drug for inhibiting labour pains while light general anæsthesia with suxamethonium will completely abolish bearing down.

### ANÆSTHESIA FOR CÆSAREAN SECTION

This is a very controversial subject. The ideal anæsthetic or analgesic should provide—

- a Good pain relief
- b Absence of respiratory depression of the foetus
- c Good relaxation of the wound
- d Absence of psychic trauma to mother
- e Absence of toxicity of mother and infant
- f Absolute safety. The foetal mortality following elective section is greater than that following normal delivery.

**Premedication**—Many workers prohibit all sedation and allow only atropine. Others allow before regional analgesia morphine  $\frac{1}{4}$  gr. believing that such a dose given within 15–20 minutes of the birth will not harm the infant. They hold that it is the dose given 2 hours before the birth which produces foetal apnoea. The injection of nalorphine into the mother or child after the mother has received a sedative may avoid foetal respiratory depression.

Nembutal or seconal  $1\frac{1}{2}$  gr. is probably free from serious effect on the child. Similarly thiopentone 0.25 g. is allowed by many if given immediately before delivery.

**General Anæsthesia**—Proper measures to prevent a piration of stomach contents must be taken (see p. 414). The anæsthetic should not be started until the surgeon is scrubbed up and ready to operate. No hypoxia must be allowed. Vomiting is frequent when the patient is in active labour. Premature infants are apt to be asphyxiated after general anæsthesia. A large stomach tube should be used if there is the slightest suspicion that the stomach may contain vomitable material.

Any general anæsthetic carefully given to lower Plane 1 or upper Plane 2 of surgical anæsthesia is suitable. After the delivery of the child anæsthesia can be deepened if necessary. The more a general anæsthetic agent is pushed to produce relaxation the more likely is the child to be born apnoeic.

General anæsthesia is preferred by the surgeon who likes a moderately soft uterine muscle to stitch up.

Good results have been claimed for thiopentone with nitrous oxide trilene and plenty of

together

e a x f

has found the following technique satisfactory. The patient premedicated with atropine 0.6 mg. is placed on the table each arm resting on an arm board. Anaesthesia is induced with thiopentone 0.2-0.25 g. and maintained with gas oxygen (70-30) and trilete. As soon as unconsciousness is reached the legs above the knees and the wrists are securely and rapidly fixed down and the incision is made. Movement is controlled by the straps and does not matter so long as analgesia is present. Very light anaesthesia is maintained until the baby is delivered and then the anaesthetist settles down to provide the surgeon with a reasonable anaesthetic and a quiet patient. At this stage intravenous thiopentone or morphine ( $\frac{1}{2}$  gr. to mg.) provides rapid and effective control and a relaxant can be given if necessary. Babies delivered in this manner breathe spontaneously and the surgeon usually co-operates with the anaesthetist during the initial rather turbulent conditions knowing that soon peace will be restored.

Atropine thiopentone 0.2 g. followed by cyclopropane and flaxedil 60 mg. will also be found to be a suitable sequence.

In all cases a high percentage of oxygen should be given to the mother immediately before the child is separated.

**Subarachnoid Block**—This is considered by many to be dangerous as an analgesic for Caesarean section. Many maternal deaths have been reported. It is a method without bad effect on the child provided that the obstetrician extracts it as rapidly as possible after the onset of analgesia. Should he dilly-dally at this time foetal hypoxia may result from the contracted uterus compressing the placenta. It ensures good retraction and absence of post partum haemorrhage. It is the safest method of providing really good relaxation should the surgeon require this. The enlarged uterus by interfering with the movements of the diaphragm tends to produce hypoxia of the mother. As Macintosh points out this hypoxia may be the cause of some of the deaths that have been reported. Certainly 100 per cent oxygen should be given to the mother from the outset. A suitable vasopressor such as methoxamine 5-10 mg. should be injected intramuscularly before the subarachnoid injection to minimize fall in blood pressure (to be maintained above 90 mm Hg).

Block should reach the T<sub>10</sub> T<sub>11</sub> margin and can be obtained with heavy nupercaine 1.4-1.6 ml. injected with the patient in the lateral position and afterwards turned on to her back on a level table. Hyperbaric amethocaine hydrochloride (8 mg. in 1 per cent solution with 1 ml. of 10 per cent dextrose) or procaine hydrochloride (100 mg. of crystals dissolved in 2 ml. of cerebrospinal fluid) can be used in the same way—all three solutions being hyperbaric. A safety measure is the use of the serial spinal technique of Lemmon in which procaine 100 mg. is the initial dose more being given if required. Supplementary anaesthesia should be withheld until the birth of the child after which a little thiopentone or inhalation anaesthetic can be given if necessary and if it is certain that the stomach is empty.

**Subarachnoid Block for Cæsarean Section** *continued*

The cocaine derivatives are said to be specially toxic to the pregnant woman nupercaine is not of course one of these Evidence has been produced to show that the circulation of the cerebro spinal fluid is altered during the later months of pregnancy perhaps because of the pressure of the tumour on the large abdominal and thoracic veins Nevertheless in spite of objections from very experienced workers subarachnoid block has gained some popularity as an analgesic method for Cæsarean section Great care is required especially in control of the blood pressure and in adequate oxygenation of the mother and hence the child An intravenous drip should be set up and frequent estimations of the blood pressure should be taken The systolic blood pressure should be prevented by the intravenous injection of a pressor drug (e.g. methoxamine 5-10 mg) from falling below 90 mm Hg

**Local Infiltration**—This is without serious effect on the mother or child but is unsuitable for frightened or uncontrolled patients The surgeon's co-operation is essential for success he usually performs the injections himself

Intradermal and subcutaneous infiltration is carried out in the line of the incision and solution should be deposited for about 1 in on each side of the midline Extra solution is injected into the pyramidales and into the retropubic space of Retzius Solution injected into the rectus sheath will improve relaxation The parietal peritoneum is infiltrated likewise the tissue overlying the lower segment if the classical operation is not to be employed Those interested in the technique should consult the paper by A C Beck (*Amer J Obstet Gynec* 1947 43 815)

Many obstetricians prefer the administration of 0.25 g of thiopentone just before the uterus is incised oxygen being given at the same time

**Muscle Relaxants**—Successful results have been obtained by the following technique With atropine as premedication kemithal 0.4 g is injected intravenously—it is stated to be less depressing to the foetal respiratory mechanism than thiopentone This is followed by *d* tubocurarine chloride 15 mg (after a trial dose) and anaesthesia is maintained with cyclopropane (Gray) Both uterine retraction and a spontaneously crying baby usually result

**Continuous Caudal Analgesia**—(See also Chapter XVIII) A method not often used in Britain If lignocaine 1-1.5 per cent solution is employed the dose should be 25-35 ml If 1 or 1½ per cent metycaine is used an average of 60-80 ml is required An intravenous drip should be set up as a routine From half to three quarters of an hour may be required for analgesia to be complete to the costal margin The blood pressure must be observed charted and controlled and oxygen 100 per cent should be given during the analgesia and operation Good results have been obtained using 30 ml of solution to use caudal

analgesia with infiltration of the line of incision with 0.5 per cent procaine

Babies born under this type of analgesia usually cry spontaneously

**Extradural Lumbar Block.**—Good results have been achieved with this method in non urgent cases. Injection is between L2 and L3 25-35 ml of 1 per cent or 1.5 per cent lignocaine. Analgesia a little above the eighth thoracic dermatome appears in five to ten minutes and may last 1½ to 2 hours. Early movement after operation is possible as motor paralysis is not complete. The continuous technique has its use here. Headaches and pareses of nerves do not occur.

This whole difficult question was discussed at the Royal Society of Medicine\* to the reports of which readers are referred.

### RESUSCITATION OF THE NEWBORN

The foetal blood haemoglobin is 15 to 20 g per 100 ml and when fully saturated carries 22 vol per cent of oxygen. But because of the low oxygen partial pressure at which maternal blood gives up its oxygen foetal haemoglobin is only 50 per cent saturated. To compensate for this hypoxia foetal haemoglobin carries more oxygen at a lower tension i.e. the dissociation curve of foetal haemoglobin is shifted to the left while the dissociation curve of the maternal haemoglobin is shifted to the right making it give up oxygen more easily. The carbon dioxide content of foetal blood is increased. The low metabolic rate of the foetus allows this hypoxia to be tolerated.

At birth the skin reflexes are ill developed except in the area supplied by the trigeminal nerve. Mouth and pharyngeal stimulation may help to excite respiration.

In the newborn the amount of carbonic anhydrase is half that found in adult blood so that release of carbon dioxide in lungs is handicapped.

During the process of birth anaerobic glycolysis may aid the survival of the infant should respiratory embarrassment occur oxygen being released from glycogen.

Intra uterine respiration of the foetus the rhythmical amniotic tide into and out of the air passages was first demonstrated by Ahlfelt in 1888 and then by the Italian Ferroni in 1899. More recently Snyder and Rosenfeld (*Amer J Physiol* 1937 119 153) have shown that maternal hypercapnia fails to stimulate foetal respiration but hypoxia inhibits it. Also foetal hypoxia fails to stimulate respiration presumably because of the absence of the aortic-carotid body reflex in the foetal and neonatal state. Hypoxia thus does not cause the initiation of respiration.

The signs of intra uterine hypoxia are irregularity of the foetal heart rate going on to tachycardia and bradycardia. In a head presentation the presence of meconium indicates hypoxic relaxation of the foetal anal sphincter.

Respiratory failure in the newborn may be

- 1 CENTRAL.—Due to (a) Immaturity of respiratory centre (b) Damage to respiratory centre from trauma (c) Oxygen

**Resuscitation of the Newborn continued**

lack (d) Narcotics and sedatives given to mother The threshold of maternal respiratory centre differs from that of the foetal centre as a level of narcosis harmless to the mother may be depressing to foetus All general anæsthetics to mother are hazardous if the baby is premature

- \* PERIPHERAL—Due to (a) Immaturity of lungs (b) Respiratory obstruction (c) Muscular weakness

The baby recovering from asphyxia first takes a series of gasps which give place to a series of single prolonged inspirations Finally rhythmic inspiration and expiration set in Periodic breathing is not of bad prognostic significance in newborn babies and is usual in premature infants

**Causes of Hypoxia in Foetus and Newborn \***—Hypoxia is of the hypoxic type it may be either sudden or continuous and prolonged and is due to —

- 1 Reduction of oxygen tension in maternal blood
- 2 The trauma of labour
- 3 Interference of passage of oxygen from mother to foetus through —
  - a Hypotension due to spinal or caudal analgesia
  - b Anæmia e.g. hæmorrhage cardiac failure etc
  - c Tetanic uterine contractions sometimes caused by pituitary extract or spinal analgesia
  - d Placental infarction or premature separation
  - e Prolapse or knotting of cord

- 4 Depression of foetal respiratory centre by narcotics sedatives and anæsthetics all of which depress it in concentrations less than those required to depress the maternal respiratory mechanism Nitrous oxide with 20 per cent oxygen is the only means of relieving the pains of labour without influencing the foetal respiratory centre Nitrous oxide with not less than 15 per cent oxygen can however be given for short three minute periods intermittently without seriously harming the foetus

Foetal distress demands oxygen to the mother There is evidence that asphyxia at birth may cause permanent cerebral or neurological damage If placental function is suspect or labour shows the placental reserve to be low e.g. if foetal heart rate falls ten beats per minute during the pains then no form of analgesia lowering the maternal blood oxygen tension should be used such as gas-air A maximal maternal blood oxygen tension should be provided also in long labours and pregnancy toxæmia

Of all anæsthetic and associated drugs used in labour only the muscle relaxants (with the possible exception of gallamine) seem clinically to treat the placental barrier as an iron curtain and keep on the maternal side of it

**Management of Asphyxia of the Newborn**—The foetus in utero is cyanosed The normally delivered child should breathe rhythmically from the beginning air replacing liquor amni as the fluid is respired Alveoli are opened up by expiration against a positive

pressure—as in crying. In respiratory depression respiration begins differently in gap—the most primitive respiratory movement involving many muscles.

Flag separates asphyxia neonatorum into three stages. (1) The stage of depression when the infant is flaccid. (2) The stage of spasticity with irregular gasping respiration, cyanosis, active upper respiratory tract reflexes, and spasticity of muscles due to hypoxia. (3) The stage of flaccidity with absent reflexes, flaccid muscles, and failing circulation.

The fundamental pathology is atelectasis and is accompanied by decrease in oxygen content and saturation, a rise in carbon dioxide tension and sometimes a fall in carbon dioxide content (due to its displacement from base by the large increase in the lactic acid in the blood).

Blue asphyxia seldom requires more than clearing the upper air passages by suction etc. using a rubber-ended rather than a metal-ended instrument. Skin stimulation, passive limb movements, slapping etc. are also very valuable while oxygen given via a nasal catheter is also beneficial. Another useful method is to insufflate 2 to 3 litres a minute of oxygen from a small rubber catheter into the infant's mouth while its mouth and nares are occluded. This is sufficient to distend the lungs and can be continued rhythmically for some time without causing trauma\*. Carbon dioxide is theoretically contra-indicated as hypercapnia is already present. Nevertheless many clinicians have faith in its respiratory stimulating properties. In feeble babies a small stomach tube should be passed to evacuate the stomach of liquor amni and prevent its aspiration into the lungs.

White asphyxia or shock (the stage of flaccidity) requires more energetic measures. If breathing does not soon commence after careful suction and clearing of the air passages the larynx should be very gently intubated via a baby's laryngoscope or by touch. A Magill tube size 00 or a rubber catheter size 3 or 4 on a wire stylet is convenient. The tube can be connected to an oxygen cylinder via a small rubber reservoir bag, a T tube and a reducing valve. The open end of the T tube allows for expiration and prevents too much pressure from being exerted. If an oxygen flow of  $1\frac{1}{2}$  litres a minute is supplied, occlusion of the open end of the T tube for three seconds ten to fifteen times a minute will allow a safe and beneficial pressure to be built up. Blaikley incorporates a water manometer into the system and this should not register in excess of 20 cm. of water. To intubate a neonate the patient should be on a flat table with no pillow. The head should not be extended over the end of the table. The blade of the infant laryngoscope should be inserted as far as the glottis and then lifted vertically. Rhythmic inflation of oxygen often has beneficial results (probably because of changes in intrabronchial pressure rather than because of expansion of collapsed alveoli) although this view is disputed by some obstetricians who deprecate this method of resuscitation.



**Resuscitation of the Newborn continued**

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\* Knowles G. S. A. *Brit. med. J.*, 1952, 2, 1551.

**Resuscitation of the Newborn continued**

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## CHAPTER XVIII

## THERAPEUTIC ASPECTS OF ANÆSTHESIA

## REGIONAL ANALGESIA

1. **Somatic Nerve Pain.**—Can be relieved by injection of local analgesic solution into suitable nerves if they are approached from the skin surface. The following are examples —  
The trigeminal nerve and its branches in patients not suitable for retro-Gasserian ganglionectomy who are suffering from tic douloureux inferior dental block in some cases of fractured lower jaw infra-orbital supra-orbital supratrochlear and mental nerve block for herpes zoster facial nerve block for intractable spasm superior laryngeal nerve block for carcinoma of larynx (see p 326) accessory nerve block for traumatic torticollis (together with superficial cervical plexus block) Injection into the temporomandibular joint in severe cases of painful clicking jaw Suprascapular nerve block for sub-acromial bursitis and pain on abduction of the arm † (See p 515) Phrenic nerve block in intractable hiccup (see p 514) Intercostal nerve block for herpes zoster post operative abdominal pain post-operative or post traumatic atelectasis fractured ribs ‡ or pain following the insertion of a drainage tube Block of lumbar nerves for intractable neuritis (see pp 366 342) herpes zoster lightning pains of tabes § Periarthritic injections for severe sprain and injections into painful amputation stumps and scars flat feet ¶ and anterior and posterior femoral cutaneous nerve block for hip joint pain || When long acting relief is required it may be necessary to employ such a drug as efocaine or alcohol in some of these patients
2. **Sympathetic Block**—Sympathetic dysfunction can cause symptoms due to vasospasm the production of pain and alteration of function so that therapy is directed to vasodilatation relief of pain and restoration of function  
Vasomotor block may be performed at any of four levels —
  - a Peripheral nerve block e.g. the ulnar nerve causing vasodilatation of the skin of the little finger
  - b The sympathetic ganglia e.g. the stellate or the second and third lumbar ganglia causing release of vasomotor tone in the upper and lower limbs
  - c Extradural block
  - d Subarachnoid block

Rollason, W. N. *Brit J Anæsth* 1955 27 354.  
† Wertheim H. M. and Rovenstin F. A. *J. Anæsth* 1941 2 541.  
‡ Pow H. H. D. W. *Brit med J* 1955 2 829.  
§ Fowler W. *Brit J Gen Pract* 1947 23 90.  
¶ Hipps H. E. and Neely H. *Na med B* 1945 44 262.  
|| Lundy J. S. and others *Proc Mayo Clin* 1951 28 281.

Management of Asphyxia of the Newborn *continued*

There is evidence that a positive pressure of 30-60 cm of water applied through an endotracheal tube for repeated short periods will cause a collapsed lung to expand.\*

Oxygen can be absorbed from the gastric mucosa as well as from that of the bronchi† and oxygen administered via a stomach tube—in the absence of anyone skilled in laryngoscopy in infants—is a useful first aid measure. Ostry‡ has devised a double tube adapted from a Miller Abbott tube for this purpose. Over inflation of the stomach is prevented. Premature babies must not be given pure oxygen. If they are cyanosed they may receive 40 per cent oxygen for short periods only thus reducing the danger of retrolental fibroplasia.

Injection into the umbilical vein of *alpha lobeline* gr  $\frac{1}{8}$  may help while 0.1 to 0.5 mg of *N-allyl nor morphine* (nalorphine lethrone) similarly injected will counteract any respiratory depression following injection of pethidine or morphine into the mother. It may have to be repeated as its stimulant effect may be shorter than the depressant effect of the sedative. Another method is to give an intravenous dose of 10 mg to the mother just before delivery if she has recently received morphine or one of its congeners. The new drug *amiphenazole* (daptazole) gives promise as a respiratory stimulant. Like nalorphine it can be given either to the mother who has recently received morphine or pethidine (20 mg intra muscularly) or into the infant's umbilical vein (3 mg). It is said to be relatively harmless and does not abolish the analgesic effects of opiates etc in the mother. Its effects are less prolonged than those of morphine in labour. It reduces but does not abolish neonatal apnoea caused by morphine.§

Electrical stimulation of the phrenic nerves through the intact skin was first applied by Hufeland in 1783 and again by Ziemssen in 1857. Sarnoff (1948) and Cross (1950) have recently reported good results following its use. Stimulation of the phrenic by a suitable electric current will cause contraction of the diaphragm and inspiration¶. It cannot cause emphysema. The phrenic motor point lies anterior to the scalenus anticus muscle and is most readily palpated if the supine patient faces directly upwards. The airway must of course be patent. The method has also been used successfully in adults.

Focal epilepsy in later life can often be ascribed to neonatal hypoxia or to improper head compression during labour||.

This whole question is well discussed in *Proc R Soc Med* 1950

43 6

Goddard R. F. C. *et Res Anæsth* 1953 34 1

† Akerrén, Y. and Furstenberg N. *J Obstet Gynec Brit Emp* 1950 57 705

‡ Ostry E. I. *Ibid* 1951 58 6

§ Holmes, J. M. *Lancet* 1956 2 765

¶ See also Cross, K. W. and Roberts P. W. *Brit med J* 1951 1 1043 and

Sarnoff S. J. *Ibid* 1951 1 1515

|| Penfield, Wilder *Curr Res Anæsth* 1954, 32, 145

pain of biliary colic while block of T 12 and L 1 will relieve renal colic. If the eighth ninth and tenth thoracic ganglia are blocked the pain of acute pancreatitis will be relieved. Retroperitoneal hematoma has followed paravertebral blocks. No blocks should be undertaken in a patient who is being treated with anticoagulants.

### INTRAVENOUS PROCAINE

(See also p. 249)

Good reports of its use which is generally in 0.2 per cent solution are reported in a large variety of conditions including —

- 1 Lower nephron nephrosis
- 2 Painful vasomotor and trophic conditions such as frost bite and intermittent claudication
- 3 Amblyopia following retinal vascular occlusion or massive hæmorrhage
- 4 Orchitis and epididymitis
- 5 To speed up intravenous drips by relaxing the vein walls
- 6 Pruritus
- 7 Serum sickness and other states of sensitivity    urticarias associated with blood transfusions
- 8 To control post-operative pain
- 9 To relieve the pain of chest injury
- 10 To relieve the pain and reduce the intra-ocular tension in acute glaucoma

**Xylocaine** has also been given with success intravenously. It has proved beneficial in the pain of general carcinomatosis and as an analgesic during labour and also during surgery.\*

### ANÆSTHETIC TECHNIQUE APPLIED TO TREATMENT IN OBSTETRICS

In eclampsia subarachnoid block up to T 8 relieves hypertension, reduces the venous return to the heart, decreases the peripheral resistance, relieves pulmonary œdema and increases the blood flow to the kidneys†. The continuous technique may be used but care must be taken to avoid hypotension. Continuous caudal analgesia also finds a place here and as only sympathetic block is needed a weak solution can be used such as xylocaine 0.25 per cent solution. Better results are however given by continuous lumbar extradural block which may have to be maintained for several days at a time‡. Blood pressure can usually be satisfactorily lowered and labour can if necessary be stimulated by a pitocin drip. The phenothiazine drugs are used in the treatment of eclampsia; they cause a lowered blood pressure, sedation and diuresis.

Uncontrollable cases of post partum hæmorrhage have been successfully treated by spinal analgesia which causes contraction of the circular fibres of the uterus and so stops bleeding. Care must be taken to avoid too much hypotension.

de Cliv. Low. G. C. Destrind J. and North J. *Anæsthesia* 1958 13 138

† Lund P. C. *Anæsthesiology* 1951 17 6

‡ Bryce-Smith R. and Williams E. D. *Lancet* 1 1241

Sympathetic Block *continued*

The last two are examples of pre ganglionic block and must extend in the case of the lower limb to the tenth thoracic segment so as to paralyse all the pre ganglionic fibres going to the limb

## INDICATIONS FOR SYMPATHETIC BLOCK —

**PAINFUL LIMBS DUE TO VASCULAR DISEASE** — Raynaud's phenomena vasospasm associated with lesions of the spinal cord e.g. poliomyelitis and some cases of pyramidal disease arteriosclerosis and thrombophlebitis obliterans chronic ulceration of the extremities embolism of major vessels thrombophlebitis erythromelalgia

**CONDITIONS DUE TO IDIOPATHIC AND POST TRAUMATIC PAIN OF LIMBS** — Causalgia amputation stump neuralgias Sudeck's atrophy

**UNCLASSIFIED CONDITIONS OF THE LIMBS** — Hyperhidrosis after embolectomy in the post hyperæmic stage of the immersion foot syndrome

**THORACIC AND ABDOMINAL DISEASE** — Angina pectoris painful aortic aneurysm bronchial asthma megacolon and cardio-spasm biliary and renal colic pancreatitis (bilateral block of T 7 8 and 9) post partum hæmorrhage eclampsia to arrest bleeding after prostatectomy to differentiate organic from functional disease of the bowel

**STELLATE GANGLION BLOCK** (see p. 326) — Has been successfully used in the treatment of recent cerebral embolism and thrombosis\* (not in cases of apoplexy e.g. if there is cervical rigidity) block should be bilateral in neonates born with hemiplegia due to birth injury in central arterial or venous occlusion of the retina e.g. in quinine amblyopia † in cases of vascular spasm of the arm for painful shoulder

## TECHNIQUE —

- 1 *Upper Extremity* — The second and third thoracic sympathetic ganglia Brachial plexus block Stellate ganglion block
- 2 *Heart and Aorta* — Paravertebral sympathetic block of the second to the fifth thoracic ganglia on the left side
- 3 *Abdomen* — Subarachnoid injection of absolute alcohol is suitable for the relief of pain below the groin and iliac crests i.e. in the lumbosacral distribution The intraspinal segmental technique can be employed in which a plastic catheter is passed into the theca its exact position being checked by radiography Hirschsprung's disease in children has yielded to high subarachnoid block (to T 5) ‡ Anuria if due to cortical ischaemia, has been improved by block to T 8 For intestinal lesions block must be to T 5 or it may be extradural paravertebral or splanchnic (Kappis) Splanchnic injection of alcohol has given relief in cases of inoperable carcinoma of the pancreas Paravertebral block of T 7 and T 8 and sometimes of T 6 and T 9 in addition will ease the

\* Lenche R. *Brit med J* 1951 1 231

† Gluck L. and Mumford, J. *Ibid* 1955 2 394

‡ Telford E. D. and Huxton H. A. *Ibid* 1948 1 837

through the nose (v) Facial weakness (vi) Weakness of neck muscles. These signs call for head down position, suction and prone position followed by tracheotomy, the insertion of a cuffed endotracheal tube and intermittent positive pressure respiration.\*†

The high tracheotomy and cuffed tube technique, provide for suction of the airways, protection of the airway from soiling and an easy route for intermittent positive pressure respiration. For transport to hospital a cuffed endotracheal tube is passed under local or general anaesthesia while portable suction apparatus, breathing machine (or manual reservoir bag and gas supply) and personnel trained in their use travel with the patient. Later a planned high tracheotomy is performed under local or general anaesthesia and a cuffed rubber or metal tube is inserted through the wound. Air one hundred per cent saturated with water vapour is used for respiration, the cuff being deflated four hourly. The ventilating machine may be required to deliver 40 or 50 litres of air per minute to the patient, the tidal air being high and the rate of respiration slow with a positive pressure in the airway of 5–15 cm. of water. Inspiration should be completed in one second, expiration and pause taking three seconds. A negative phase lasting throughout the period of expiration and pause may be beneficial and does not cause acute pulmonary oedema.

Care must be taken to see that ventilation is neither too great nor too small and laboratory aid is required. Under ventilation may cause (i) A rising blood pressure (ii) Tachycardia (iii) A cold moist skin (iv) Anxiety.

A gas meter should be available to measure the respiratory minute volume. Over ventilation causes alkalosis, under ventilation acidosis. A plasma bicarbonate value of less than 20 ml g/l suggests hyperventilation and so does tetany. If the carbon dioxide in the expired air is between 3.5 and 4 per cent ventilation is probably adequate but the alveolar carbon dioxide level and the plasma pH estimation may be required. Another guide if the kidneys are healthy is the pH of the urine. If this is less than 6 it suggests carbon dioxide retention. Over ventilation will probably be followed by a pH greater than 7. Gases must be humidified to prevent the formation of crusts in the air passages. Atelectasis must be prevented or treated.

## TETANUS

Tetanus presents some problems very similar to those seen in poliomyelitis. There may be pharyngeal and laryngeal paralysis and respiratory insufficiency due to spastic not flaccid muscles. It has been treated by intermittent positive pressure respiration via a cuffed endotracheal tube through a high tracheostome with a tubocurarine‡

\* C. Supt. n. Smith, A. Spalding, J. M. H. and R. Ssell, W. Ritchie, *Lancet* 1954 1 939.  
† R. Ssell, W. Ritchie, *British Medical Journal* 1955 98.  
‡ La. se. H. C. A. *Lancet* 1953 1 37.  
§ C. Supt. n. Smith, A. Spalding, *Lancet* 1956 2 55.



## RESPIRATORY INSUFFICIENCY

The anæsthetist has a most useful part to play along with his colleagues in the management of patients suffering from respiratory insufficiency. Causes of respiratory failure—other than anæsthesia \*—

- 1 Organic disease of the central nervous system —
  - a Bulbospinal poliomyelitis
  - b Encephalitis lethargica
  - c Polyneuritis
  - d Brain stem hæmorrhage
  - e Motor neurone disease
- 2 Disease involving muscles —
  - a Myasthenia gravis
  - b Dystrophia myotonica
- 3 Poisoning by —
  - a Narcotics
  - b Coal gas
  - c Strychnine
  - d Anticholinesterases
- 4 Head injury
- 5 Chest disease and chest surgery —
  - a Emphysema
  - b Kyphoscoliosis
  - c Cor pulmonale—all may be made worse by infection asthma respiratory sedatives and obesity
- 6 Tetanus

See also The Treatment of Respiratory Inadequacy by Woolmer  
*R. Brit med Bull* 1958 14 1 54

## POLIOMYELITIS

Thus may be (a) Spinal (b) Bulbar (c) Bulbospinal

- a Spinal poliomyelitis may cause respiratory paralysis but gives rise to no trouble with upper respiratory tract secretions and can be treated in a cabinet respirator (iron lung) or in mild or recovering cases in a cuirass respirator. When the vital capacity is less than 50 per cent of normal such an appliance should be used.
- b Bulbar poliomyelitis patients can breathe but cannot maintain the integrity of their upper air passages. The semi-prone head-down position together with suction will keep such patients from harm. Intubation is not necessary.
- c Bulbospinal poliomyelitis is the dangerous type especially if the bulbar component is not noticed at the beginning of treatment in the cabinet respirator. A patient lying supine in such a respirator will if he vomits (a likely event in this illness) have the vomitus drawn into his lungs by the pressure changes induced by the machine.

Signs of bulbar involvement are (i) A rattle in the throat (ii) Dysphagia (iii) Dysphonia (iv) Regurgitation of fluids

See good articles by Woolmer R. W. *Proc. World Congress of Anaesthesiologists* 1954 9  
 Minneapolis Burgess Publishing Co. Lassen, H. C. A. *Lancet* 1953 1 57 & d R. M. W. R. *Poliomyelitis* 1956 London Arnold

through the nose. (v) Facial weakness. (vi) Weakness of neck muscles. These signs call for head down position, suction and prone position followed by tracheostomy, the insertion of a cuffed endotracheal tube and intermittent positive pressure respiration.

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C mpt, S ith A Sp Id g J M I and Ru II W Rit hi La et 1954 1 939  
† Russell W Rit h B I m d J 1955 98  
‡ Lassen H C A La et 1953 1 37  
§ C pto S ith A and oth La et 1962 50

**Tetanus continued**

or suxamethonium\*. Chloral hydrate or other sedatives or nitrous oxide-oxygen can be used to produce sedation. Good reports have also followed a continuous mephanesin drip with spontaneous respiration†

Yet another method of treating tetanus is by the injection of the phenothiazine derivatives sometimes combined with gas and oxygen anaesthesia‡. This is said to control the convulsions and virtually to eliminate the problem of secretions in the chest. The patient breathes spontaneously and no relaxants are required. A dose of 25 mg each of chlorpromazine, promethazine and pethidine is given when required.

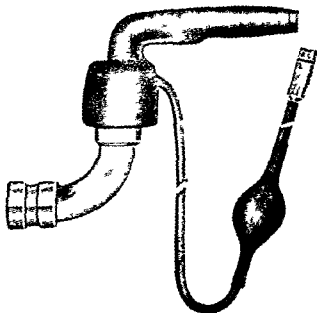


Fig. 2—Radlett rebreather tube (Medical and Industrial Equipment Ltd.)

**ACUTE PULMONARY ŒDEMA**

(See also p. 504)

This type of acute heart failure has been successfully treated by anaesthetists in consultation with their medical colleagues. The causes may be (1) Cardiac failure (2) Increased intracranial pressure (3) Inhalation of toxic gases (4) Positive intrapleural pressure

Shkilto, I. *Lancet* 1954, 2, 35

† Docherty, D. F. *Ibid.* 1955, 7, 43

Docherty, R. I., Mort, N., H. J. V. and Thomas, F. T. *Ibid.* 1955, 2, 230

(5) Pheochromocytoma (6) Hypoxia due to respiratory obstruction. The following methods have been used (1) The vicious circle can be broken by a dose of morphine or by the production of unconsciousness by a small dose of thiopentone while oxygen inhalations are given (2) An intra or extradural analgesic produces a bloodless phlebotomy the venous return to the right heart is reduced while the peripheral resistance is decreased and the burden on the left heart is eased (3) Benefit results from oxygen given under positive pressure during inspiration. This reduces the effort of breathing and so lessens the abnormally high negative intrathoracic pressure the positive pressure of the oxygen also counteracts the elevated hydrostatic pressure in the pulmonary capillaries retards blood flow to the right heart and so relieves pulmonary congestion (4) Hexamethonium by reducing the pulmonary arterial pressure relieves transudation of fluid into the alveoli. Suction should be employed in this condition whatever its cause.

### TRACHEOBRONCHIAL TOILET

This together with bronchoscopy can often be a life saving measure when carried out in the medical wards. Many patients who have the death rattle die from frank asphyxia. In selected cases adequate suction will produce a dramatic improvement in these moribund patients and if there is any vitality at all in the myocardium temporary recovery will follow.

## CHAPTER XXXIII

### POST-OPERATIVE RECOVERY ROOM\*

These rooms which should be close to the operating theatre and supervised by members of the department of anaesthesia serve a most useful purpose. Respiratory and circulatory depression are detected early and efficiently treated by the skilled nursing sister in charge of the room which is suitably equipped with oxygen therapy appliances intravenous drip apparatus beds which can be easily tipped proper lighting and suction apparatus.

The advantages of such a room are obvious. It prevents duplication of equipment economizes skilled nursing staff and saves lives. It must be remembered in this connexion that almost half the deaths occurring in the immediate post operative period are due to inadequate nursing care together with respiratory obstruction. Such a room should be staffed for twenty four hours each day one bed being sufficient for one operating theatre. Sex differentiation is unnecessary as by the time the patient is sufficiently conscious of his or her sex the time has come for transference to the ordinary ward.

*Jolly Ch and Lee J A J at the 1957 12 49 See also discussion in Proc R Soc Med 1958 51 15*

*Tetanus continued*

or suxamethonium\*. Chloral hydrate or other sedatives or nitrous oxide-oxygen can be used to produce sedation. Good reports have also followed a continuous meperidine drip with spontaneous respiration†

Yet another method of treating tetanus is by the injection of the phenothiazine derivatives sometimes combined with gas and oxygen anaesthesia. This is said to control the convulsions and virtually to eliminate the problem of secretions in the chest. The patient breathes spontaneously and no relaxants are required. A dose of .5 mg each of chlorpromazine promethazine and pethidine is given when required.

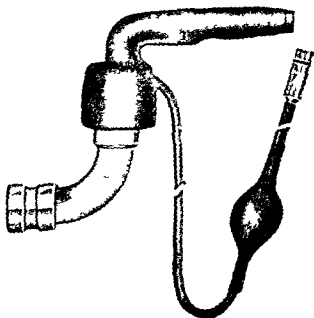


Fig. 2—Rediffusion rebreathing circuit (Med. Industrial Equipment Ltd.)

**ACUTE PULMONARY OEDEMA**

(See also p. 504)

This type of acute heart failure has been successfully treated by anaesthetists in consultation with their medical colleagues. The causes may be (1) Cardiac failure (2) Increased intracranial pressure (3) Inhalation of toxic gases (4) Positive intrapleural pressure

\* Shackleton, P. *Lancet* 1954, 2, 155.

† Docherty, D. F. *J. Med.* 1955, 1, 437.

‡ Lodman, R. I. *Anaesthesia* 1954, 9, 130. E. T. *ibid.* 1955, 2, 130.

CYLINDER DATA FOR THE MOST GENERALLY USED TYPES IN ANÆSTHESIA AND OXYGEN THERAPY  
*Cyclopropane*

Usual Description	Average Weight of Empty Cylinder	Approximate Dimensions	Weight of Gas	Equivalent in Gallons at 15 C. and 760 mm Hg	Equivalent in Litres at 15 C. and 760 mm Hg	Equivalent in Cubic Feet at 15 C. and 760 mm Hg	Usual Valve Type	Colour (B.S.I.)
gallons 8	1½	in. 0-5 x 0-1½	oz 2½	8	56	1.3	Flush Type	Orange
0	3	0-9 x 0-2½	5½	20	9½	3.2	"	"
40	5½	1-0 x 0-3	11½	40	18½	6.4	"	"
50	9½	1-4 x 0-3½	22½	80	36½	12.7	"	"

*Nitrous Oxide*

Usual Description	Average Weight of Empty Cylinder	Approximate Dimensions	Weight of Gas	Contents in Gallons at 15 C. and 760 mm Hg	Contents in Litres at 15 C. and 760 mm Hg	Contents in Cubic Feet at 15 C. and 760 mm Hg	Usual Valve Type	Colour (B.S.I.)
gallons 25	3	in. 0-9 x 0-2	lb 0 7½	25	114	4	7 or 8	French blue
50	5½	1-0 x 0-3	0 15	50	227	8	"	"
100	9½	1-4 x 0-3½	1 14	100	455	16	"	"
200	16	1-8 x 0-4	3 12	200	909	32	"	"
400	22	2-10 x 0-4	7 8	400	1818	64	"	"
500	33	3-1 x 0-4½	9 6	500	2273	80	"	"
800	45	3-0 x 0-5½	15 0	800	3637	128	"	"
2000	118	4 6 x 0-7	37 8	2000	9092	320	"	"

## APPENDIX

CONVERSION TABLE FOR THE MORE GENERALLY USED  
SIZES OF NEEDLES

SIZE	DIAMETER		LENGTH	
	mm.	s w g	mm	n
19	45	26	17.5	$\frac{1}{16}$
18	43	26	19	$\frac{1}{8}$
17	50	25	23.5	$\frac{1}{4}$
16	55	24	25	$\frac{1}{2}$
15	60	23	25	1
14	60	23	30	1 $\frac{1}{2}$
12	65	23	30	1 $\frac{1}{2}$
2	70	22	33	1 $\frac{1}{2}$
1	80	21	38	1 $\frac{1}{2}$
0	90	20	41.5	1 $\frac{1}{2}$

## DURATION OF CYLINDER CONTENTS

100 Gallons Nitrous Oxide—455 Litres

At	1 litre per minute will last	7.500 hours
2		3.750
3		2.500
4		1.875
5		1.500
6		1.250
7		0.937

33 Gallons Oxygen—150 Litres

At	200 c.c. per minute will last	12.47 hours
400		6.23
600		4.15
800		3.12
1 litre		2.49
2		1.25
3		0.83
4		0.62

110 Cu Ft Oxygen—3119 Litres

At	20 litres per minute will last	25.96 hours
25		20.76
30		17.30
35		14.84
40		12.98
45		11.52
50		10.38
55		9.44
60		8.65
65		7.98
70		7.42
75		6.92
80		6.49

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Some Normal Biochemical Values *continued*2. *Urine* —

Reaction	pH 4.8-7.4
Specific gravity	1.010-1.025
Daily output	1000-1500 c.c.

## CONCENTRATIONS OF ELECTROLYTES IN PLASMA

Sodium	142 milliequivalents per litre
Potassium	5
Calcium	5
Magnesium	3
Bicarbonates	27 milliequivalents per litre
Chlorides	103
Phosphates	2
Sulphates	1
Protein	16

## CONCENTRATION OF ELECTROLYTES IN URINE

Sodium	100-250 milliequivalents in 24 hours
Potassium	35-90
Chloride	170-550
Phosphates	30-90

1 milliequivalent of chloride = 35.5 mg

1 milliequivalent of sodium = 23 mg

## PULSE AND RESPIRATION RATE OF CHILDREN

	Pulse-rate	Respiration rate
Birth	140-170	50-70
1st yr	125-165	35-55
2-5 yr	105-90	25
5-14 yr	85-75	25-20

Systolic blood pressure 75-90 mm. Hg in infancy

## SOLUTIONS

A *molar solution* contains the molecular weight of a substance expressed in grammes dissolved in 1 litre

A *milliequivalent per litre (mEq/l)* is the equivalent weight in grammes per litre divided by 1000. To convert milligrammes per cent into milliequivalents per litre (mEq/l) divide the concentration in mg per cent by the atomic weight of the substance and multiply by its valency

## Strength of Solution —

1:4000 = 0.025 per cent	1:1000 = 0.05 per cent	1:333 = 0.075 per cent
1:1000 = 0.1 per cent	1:666 = 0.15 per cent	1:500 = 0.2 per cent
1:400 = 0.25 per cent		



Some Normal Biochemical Values *continued*2. *Urine* —

Reaction	pH 4.8-7.4
Specific gravity	1.010-1.025
Daily output	1000-1800 c.c.

## CONCENTRATIONS OF ELECTROLYTES IN PLASMA

Sodium	142 milliequivalents per litre
Potassium	5
Calcium	5
Magnesium	3
Bicarbonates	27 milliequivalents per litre
Chlorides	102
Phosphates	2
Sulphates	1
Protein	16 "

## CONCENTRATION OF ELECTROLYTES IN URINE

Sodium	100-250 milliequivalents in 24 hours
Potassium	35-90
Chloride	170-250
Phosphates	30-90

1 milliequivalent of chloride = 35.5 mg

1 milliequivalent of sodium = 3 mg

## PULSE AND RESPIRATION RATE OF CHILDREN

	Pulse-rate	Respiration rate
Birth	140-170	50-72
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2-6 yr	105-90	25
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1:1000 = 0.1 per cent	1:666 = 0.5 per cent	500 = 0.5 per cent
per cent		400 = 0.25 per cent

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Some Normal Biochemical Values *continued*

## 2 Urine —

Reaction	pH 4.5-7.4
Specific gravity	1020-1025
Daily output	1000-1800 c.c.

## CONCENTRATIONS OF ELECTROLYTES IN PLASMA

Sodium	142 milliequivalents per litre
Potassium	5
Calcium	5
Magnesium	3
Bicarbonates	27 milliequivalents per litre
Chlorides	102
Phosphates	2
Sulphates	1
Protein	16

## CONCENTRATION OF ELECTROLYTES IN URINE

Sodium	100-150 milliequivalents in 24 hours
Potassium	35-90
Chloride	170-250
Phosphates	30-90

1 milliequivalent of chloride = 35.5 mg

1 milliequivalent of sodium = 23 mg

## PULSE AND RESPIRATION RATE OF CHILDREN

	Pulse rate	Respiration rate
Birth	140-120	50-32
1st yr	115-105	33-25
2-6 yr	95-90	25
7-14 yr	85-75	23-20

Systolic blood pressure 75-90 mm Hg in infancy

## SOLUTIONS

A *molar solution* contains the molecular weight of substance dissolved in litres

A *milliequivalent per litre (mEq/l)* is the equivalent weight in grammes per litre divided by 1000. To convert milligrammes per cent to milliequivalents per litre (mEq/l) divide the concentration in milligrammes per cent by the atomic weight of the substance and multiply by its valency

## Strength of Solution —

1-4000 = 0.025 per cent      1000 = 0.05 per cent      1333 = 0.025 per cent  
 1-1000 = 0.1 per cent      1-666 = 0.15 per cent      100 = 0.1 per cent      400 = 0.25 per cent

[illegible]

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